COMPARATIVE EFFICACY OF WEEKLY ONCE IRON WITH AND WITHOUT VITAMIN C SUPPLEMENTATION IN ANAEMIC ADOLESCENT GIRLS

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CERTIFICATE

Certified that this dissertation entitled **"COMPARATIVE EFFICACY OF WEEKLY ONCE IRON WITH AND WITHOUT VITAMIN C SUPPLEMENTATION IN ANAEMIC ADOLESCENT GIRLS"** *is a bonafide work done by* ***DR.S.STALIN,*** *post graduate student of Paediatric Medicine, Kilpauk Medical College Hospital, Chennai – 10, during the academic year 2009-2011.*

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DECLARATION

I declare that this dissertation entitled ***"COMPARATIVE EFFICACY OF WEEKLY ONCE IRON WITH AND WITHOUT VITAMIN C SUPPLEMENTATION IN ANAEMIC ADOLESCENT GIRLS"*** *has been conducted by me at Kilpauk Medical College Hospital. It is submitted in part of fulfillment of the award of the degree of M.D., (Paediatrics) for the April 2011 examination to be held under the Tamil Nadu DR.M.G.R Medical University, Chennai. This has not been submitted previously by me for the award of any degree or diploma from any other university.*

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CONTENTS

INTRODUCTION

Nutritional deficiency disorders constitute a major health problem in India. In addition to direct implications for morbidity and mortality, undernutrition predisposes children and adults to various infections. Three micronutrients – vitamin A, iron and iodine are among the most important of all the nutrients needed by the body because they are vital for developing normal learning and cognitive functions, immune defence mechanism, work capacity and reproductive health[1]. Deficiency of these three micronutrients is known to have devastating effects on health.

Anemia resulting from lack of sufficient iron for the synthesis of hemoglobin is the most common hematological disease of infancy and childhood[21]. It is estimated that 30% of the global population suffers from iron deficiency anemia; most of those affected live in developing countries[21].

Iron deficiency anemia occurs when iron absorption cannot compensate iron requirements and losses. Requirements are especially high in pregnant women, infants, young children and adolescents. The consequences of iron deficiency anemia are many and serious, affecting not only individual health but also the development of societies and countries.[46]

In India, the prevalence of anaemia among adolescent girls is high. Adolescent girls are particularly vulnerable group as their requirements of iron as well as its losses from the body are high. And the bioavailability of

iron is also low in predominantly cereal based diets because of their high phytate content. Anemia is likely to compromise physical work capacity and cognitive functions of girls in the pubertal phase of development[43]. Anemia during adolescence limits growth and delays the onset of menarche, which inturn may later lead to cephalopelvic disproportion. It results in increased maternal mortality and decreased child survival, as supplementation during pregnancy fails to restore the iron status.

Very often, in India, girls get married and become pregnant even before the growth period is over, making anemia doubly risky. Few programmes for anemia control have targeted adolescent girls and health care of adolescent girls all over the world has not been given priority.

Global Scenario [4-7]

Iron deficiency anemia affects nearly 2 billion people around the world. Of these nearly 90% are in the developing countries.

Indian Scenario [4-7]

In India according to NSHF survey, 74% of children, 32 – 55% of adolescent girls and 50 – 60% of pregnant women are affected by iron deficiency anemia. 5.8 – 48.7% of pregnant women are severely anemic. 19% of maternal death is attributed to iron deficiency anemia alone.

Tamil Nadu Scenario [4-7]

In Tamil Nadu 69% of children and 44.8% of adolescent girls are affected by iron deficiency anemia. Though it is less than national level, it is more than the desired level.

Anemia[21]

Anemia is defined as a reduction of the red blood cell (RBC) volume or hemoglobin concentration below the range of values occurring in healthy persons. Although a reduction in the amount of circulating hemoglobin decreases the oxygen-carrying capacity of the blood, few clinical disturbances occur until the hemoglobin level falls below 7–8 g/dl. Below this level, pallor becomes evident in the mucous membranes. When moderately severe anemia develops slowly, surprisingly few symptoms or objective findings may be evident. Weakness, tachypnea, shortness of breath on exertion, tachycardia, cardiac dilatation, and congestive heart failure ultimately result from increasingly severe anemia, regardless of its cause. Anemia is not a specific entity but, rather, the result of many underlying pathologic processes.

Anemia with adequate response (Reticulocyte Production Index >3)	
1) Hemoglobinopathy	Hemoglobin SS,S-C, Sβ thalassemia
2) Enzymopathy	G6PD def, pyruvate kinase def.
3) Membranopathy	Hereditary spherocytosis, elliptocytosis, ovalocytosis
4) Extrinsic factors	DIC, HUS, TTP, Burns, Vitamin E def., Abetalipoprotenemia, Wilson disease
5) Immune hemolytic anemia	Autoimmune, isoimmune, drug induced

Anemia with inadequate response (Reticulocyte Production Index<2)		
Microcytic hypochromic	**Normocytic normochromic**	**Macrocytic**
1)Iron deficiency 2)Thalassemia 3)Chronic inflammatory disease 4)Copper deficiency 5)Sideroblastic anemia 6)Aluminium , lead intoxication 7)Hereditary pyropoikilocytoses 8)Hemoglobin CC,EE	1)Chronic inflammatory dis. 2)Recent blood loss 3)Malignancy/marrow infiltration 4)Chronic renal failure 5)Transient erythro-blastopenia of childhood 6)Marrow aplasia/hypoplasia 7)HIV infection 8)Hemophagocytic syndrome	1)Vitamin B12 def. 2)Folate def. 3)Hypothyroidism 4)Marrow failure 5)Orotic aciduria 6)Chronic liver disease 7) Lesch Nyhan syndrome 8) Downs syndrome 9) Drugs

A brief overview of the iron metabolism and iron deficiency anemia will help in understanding alternate strategies.

Iron Metabolism and iron deficiency Anemia[17, 21]

Iron deficiency anemia is the most common anemia in the world. The fourth most abundant element in the earth's crust, iron is only trace element in biologic systems, making up only 0.004% of the body's mass. Yet it is an essential component or cofactor of numerous metabolic reactions. By weight, the great proportion of the body's iron is dedicated to its essential role as a structural component of hemoglobin. Without sufficient iron available to the RBC precursors, normal erythropoiesis cannot take place, and anemia develops. On the other hand, iron is a toxic substance. Too much iron accumulating in vital structures (especially the heart, pancreas, and liver) produces a potentially fatal condition, hemochromatosis. Iron is an essential component of the hemoglobin molecule, without iron the marrow is unable to produce hemoglobin. The red cell number falls and those which do reach the circulation are smaller than normal (microcytic) and lack hemoglobin, hence they are pale and under coloured (hypochromic). The deficiency in iron may be absolute, that is, there is no iron available for the production of hemoglobin, and this is true iron deficiency anemia. The deficiency may be relative, that is, the iron is present in storage form in the

marrow but is unavailable for hemoglobin production, and this is anemia of chronic disease.

Iron distribution

The majority of the iron is present as hemoglobin iron. Approximately 25% of the iron is maintained as storage iron (ferritin and hemosiderin) primarily in the bone marrow.

-Hemoglobin -1.5 to 3.0gm (65 to 70%)

-Storage

Ferritin & Hemosiderin -0.5 to 1.5gm (20 to 30%)

-Others

Myoglobin

Heme enzyme remainder

Iron absorption[48]

Iron absorption occurs primarily in the duodenum. Most of the iron is in the ferric (+++) form and is complexed to other organic and inorganic molecules. The acid in the stomach and hydrolytic enzymes in the small intestine release the iron from these complexes. It is then reduced to the ferrous (++) form as it is more readily absorbed in this state. Absorption is increased by the presence of glucose, some amino acids, ascorbic acid (Vitamin C).

These substances aid in the absorption process by either reducing ferric iron to the ferrous state or by helping bind the iron to the mucosal cell receptor sites. Heme iron, iron in meat myoglobin, is more easily absorbed than elemental iron. Iron absorption is decreased by the presence of phosphate, bicarbonate, bile acids, phytates.

Fe^{3+} reductase activity is associated with the iron transporter in the brush borders of the enterocytes . Once the ferrous iron (++) binds to receptors on the surface of mucosal cells it is moved into the cell via DMT1. Some is stored in ferritin, and the remainder is transported out of enterocytes by a basolateral transporter named ferroportin 1. A protein called hephaestin (Hp) is associated with ferroportin 1. It is not a transporter itself, but it facilitates basolateral transport. In the plasma, Fe^{2+} is converted to Fe^{3+} and bound to the iron transport protein transferrin. This protein has two iron-binding sites. Bound to transferrin the iron is transported to the marrow for use or storage. Normally, transferrin is about 35% saturated with iron, and the normal plasma iron level is about 130mcg/dl in men and 110mcg/dl in women.

Heme binds to apical transport protein in enterocytes and is carried into the cytoplasm. In the cytoplasm, HO2, a subtype of heme oxygenase, removes Fe^{2+} from porphyrin and adds it to the intracellular Fe^{2+} pool. Apoferritin is a globular protein made up of 24 subunits. Iron forms a

micelle of ferric hydroxyphospate, and in ferritin, the subunits surround this micelle. Ferritin is readily visible under electron microscope and has been used as a tracer in studies of phagocytosis and related phenomena. Ferritin molecules in lysosomal membranes may aggregate in deposits that contain as much as 50% iron. These deposits are called hemosiderin.

Role of VitaminC in iron and folic acid absorbtion[49]

Vitamin C helps in the intestinal absorption of iron. It does this by keeping the iron in a form that is more easily absorbed. Further, after the iron is absorbed, Vitamin C apparently aids in the transfer of iron from a protein that transports it in the body to a protein that stores it until it is needed. Certain enzymes (specialized proteins that keep the body's metabolic machinery running) require iron to function properly. Several of these iron-containing enzymes will not work unless Vitamin C is present. Folic acid deficiency can result in anemia. Vitamin C has been reported as necessary in the conversion of folic acid to the form of this vitamin that is active in the body.

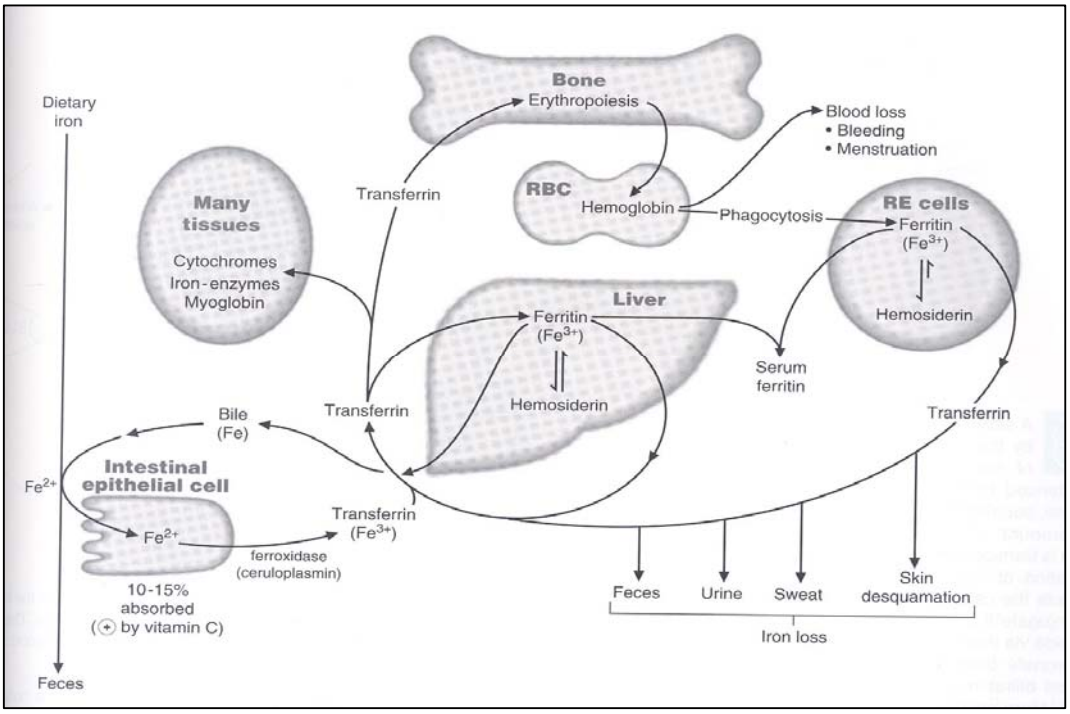
Regulation of iron absorption

The intestinal walls covered with villi, finger-like projections, which are covered with absorptive mucosal cells. These cells are produced in the crypts of Lieberkuhn, at the base of the villi, and move upwards to the villus tip to be desquamated (lost). Each cell is produced with a set amount of

apoferritin. The more iron required by the body the less apoferritin manufactured in each cell. In other words, it is the amount of apoferritin within each mucosal cell which acts as the gatekeeper and regulates the amount of iron absorbed.

Intestinal absorption of iron is regulated by three factors: recent dietary intake of iron, the state of the iron stores in the body, the state of the erythropoiesis in the bone marrow. However, the ways these factors signal the absorptive process still unsettled.

Transferrin is the primary iron transport protein. It is a beta globulin and is produced in liver. It has a half- life of 8-11days. Each molecule of transferrin can bind and transport two molecules of iron in the ferric (+++) state. Transferrin prefers to carry iron to the marrow but will carry iron to the other organs if the marrow is damaged or excess amounts of iron are already stored in the marrow. In rare instances when transferrin is absent (atransferrinemia) other proteins can bind iron but carry the iron to the other organs such as liver, spleen and pancreas, little if any is carried to the marrow. As well as specific receptors for iron, transferrin has specific receptors for sites on the developing normoblast. Once bound to the cell membrane, the transferrin changes shape and releases the iron. It then returns to the portal circulation to bind more iron.



Iron transfer across the Red Cell membrane

Iron can be transferred to developing red cells either bound to transferrin or presented as ferritin to the developing cells as they cluster around "nurse cell" RE cells. The iron is moved into the developing red cell. Clusters of normoblast around a nurse cell are called a "feed island".

Iron Storage

Iron is stored as either ferritin or hemosiderin: ferritin consists of an outer protein shell with iron complexed within the core. The outer shell consists of 22 apoferritin molecules while the core consists of an iron/phosphate complex consisting of 4,000 to 5,000 molecules of iron in

each core. Ferritin is water soluble and a very small amount dissolved in plasma.

The ferritin reference range for males is 15-200ng/ml, 12-150 ng/ml for females and for children 6 months to 15 years is 7-140ng/ml. Ferritin is not visible by light microscopy, nor is stained by the Prussian blue reaction. Hemosiderin is aggregated ferritin molecules. The protein shell has been altered and as a result it is water insoluble. It can be seen by light microscopy as gold brown granules and is demonstrated by the Prussian blue stain.

Daily Iron Requirements

The adult male requires approximately 1mg/day, just enough to cover normal iron loss. The adult female requires approximately 2mg/day, enough for daily loss and menstruation. Pregnant females require approximately 3mg/day, enough for normal, ongoing loss and fetal requirements. Children require approximately 2mg/day, enough for normal loss and extra to produce some residual iron stores and allow for increasing red cell mass.

Causes of Iron Deficiency

- Diet –uncommon except in children
- Failure to absorption iron
- Increased utilization
- Pregnancy

- Adolescent growth
- Atransferrinemia
- Failure to utilize
- Lead poisoning
- Chronic diseases
- Blood loss

Chronic blood loss is an important cause of iron deficiency anemia particularly in older children[17].

Development of iron deficiency anemia

It must be remembered that anemia in deficiency develops slowly. Only 50% of people with iron deficiency develop iron deficiency anemia since this is a late manifestation of chronic iron deficiency. The type and severity of the anemia varies with time.

Development Stages

1. Depletion of iron stores, decreased ferritin levels, no anemia.
2. Increased transferrin levels, no anemia.
3. Fall in serum iron, no anemia.
4. Development of normocytic, normochromic anemia.
5. Development of microcytic, hypochromic anemia.

The clinical manifestation may vary with age, degree and rapidity of onset and other factors. Mild anemia is often asymptomatic. The main

symptoms are exercise dyspnoea, fatigue, palpitation, pica (consumption of substances such as ice, starch or clay, frequently found in iron deficiency anemia), syncope (particularly following exercise) and bounding pulse. Dizziness, headache, syncope, tinnitus or vertigo, irritability, difficulty in sleeping or concentrating is more frequent in severe chronic anemia. Plasma ferritin level was significantly lower in children with first febrile seizure suggesting a possible role for iron insufficiency in first febrile seizures.

In the period of later school age and early adolescence, compromised pubertal growth spurt, reduced physical work capacity and cognitive function, poor scholastic performance and behavioral disturbances.

Common signs are pallor (color of skin, oral and conjunctival mucous membrane and nail beds), tachycardia, ejection systolic murmur, mild peripheral edema and venous hum and wide pulse pressure. In old people, angina pectoris can be an important clinical manifestation.

Cardiovascular Adaptations in Anemia:

The main consequence of anemia is tissue hypoxia. If anemia has developed rapidly, there may not be adequate time for compensatory adjustments to take place, so there is sudden marked contraction of intravascular volume, resulting in postural hypotension, fall in cardiac output, shunting of blood from skin to central organs, sweating, restlessness, thirst and air hunger. If anemia occurs slowly, many adaptations occur for

the oxygen maintenance, such as increasing of plasma volume and right shift of the oxygen-hemoglobin dissociation curve.

Laboratory Diagnosis of Iron Deficiency

Routine procedures:

Hb, Hct, and RBC count are all decreased. The degree of decrease depends upon the length of time the marrow has been without sufficient supplies of iron. It must be remembered that at any stage the red cell number will not be proportionately as low as is the Hb and Hct. This is due to the fact that the marrow can continue to produce cells which are deficient in hemoglobin.

Methods used to estimate Hemoglobin are[12]:

1. Direct cyanmethemoglobin method- drabkins solution added to the blood results read by spectrophotometry-Gold standard
2. Indirect cyanmethemoglobin method- blood collected in whatman filter paper and dried after that drabkins solution added and the results analysed.
3. Hemocue method-blood collected in microcuvette directly and analysed in spectrophotometer, used in field surveys and in remote areas.

Hematologic Values During Infancy and Childhood[21]

	Hemoglobin(g/dl)		Hematocrit(%)		Reticulocytes(%)	MCV(fl)
AGE	Mean	Range	Mean	Range	Mean	Lowest
Cord blood	16.8	13.7–20.1	55	45–65	5.0	110
2 wk	16.5	13.0–20.0	50	42–66	1.0	
3 mo	12.0	9.5–14.5	36	31–41	1.0	
6 mo–6 yr	12.0	10.5–14.0	37	33–42	1.0	70–74
7–12 yr	13.0	11.0–16.0	38	34–40	1.0	76–80
Adult Female	14	12.0–16.0	42	37–47	1.6	80
Adult Male	16	14.0–18.0	47	42–52		80

Special procedures:

A bone marrow examination is seldom, if ever, performed or needed for the diagnosis of an iron deficiency anemia. If however, a bone marrow is performed the following results would be present.

Bone Marrow:

Cellularity – normal to increased.

Morphology - normoblastic with some dyserythropoiesis. Ragged reduced cytoplasm, vacuoles, multinuclearity, karyorrhexis, nuclear budding, abnormal mitosis. All these may be seen but are not the predominant features of the marrow. Iron stain - absent. The absence of iron is considered to be the “gold standard” for the diagnosis of iron deficiency.

The other findings are:

Siderocytes –absent

Serum iron –decreased

TIBC – increased.

% saturation – decreased.

Ferritin – decreased.

Free erythrocyte protoporphyrin (FEP) – increased.

Treatment

Iron supplementation is the main stay of treatment. A daily total dose of 4–6 mg/kg of elemental iron in 3 divided doses provides an optimal amount of iron for the stimulated bone marrow to use[21]. The response is monitored with reticulocyte count, hemoglobin and hematocrit.

Responses to iron therapy in iron deficiency anemia.[21]

Time after iron administration	Response
12-24 Hrs	Replacement of intracellular iron enzyme subjective improvement, decreases irritability, increases appetite
36-48 Hrs	Initial bone marrow response: erythroid hyperplasia
48-72 Hrs	Reticulocytosis, peaking at 5-7days
4-30 days	Increase in hemoglobin levels
1-3 Months	Repletion of iron stores

Iron treatment should be continued for 8 weeks after blood values are normal to replace the stores.

Failure to respond may be due to:

1. continued bleeding
2. failure to take iron
3. wrong diagnosis
4. mixed deficiency
5. other causes – inflammation
6. malabsorption – unlikely

VITAMIN C[47]

Vitamin C (ascorbic acid) is water-soluble vitamin. It is the most sensitive of all vitamins to heat. Man, monkey and guinea pig are perhaps the only species that require vitamin C in their diet.

Functions are:

1. Potent antioxidant

2. Needed for the formation of collagen and collagen provides a supporting matrix for the blood vessels and connective tissue, and for bones and cartilage.

3. Facilitates absorption of iron from vegetables by reducing ferric iron to ferrous iron.

4. It inhibits nitrosamine formation by intestinal mucosa.

Sources are:

The main dietary sources are fresh fruits and green leafy vegetables. Traces of vitamin C occur in fresh meat and fish but scarcely in cereals. Germinating pulses contain good amounts. Roots and tubers contain small amounts. Amla or the Indian gooseberry is one of the richest sources of vitamin C both in fresh as well as in the dry condition. Guavas are another cheap but rich sources of vitamin C.

Deficiency leads to:

Scurvy, signs of which are swollen and bleeding gums, subcutaneous bruising or bleeding into the skin or joints, delayed wound healing, anemia and weakness.

Requirement:

The estimated requirement for vitamin C has been recently raised from 40 to 60mg with much larger doses advocated by some.

Strategies to prevent iron deficiency anemia

The major factors responsible for iron deficiency anemia in developing countries are reduced intake and poor bioavailability of dietary iron. The iron requirements in different age groups are different.

WHO expert group proposed that anemia should be considered to exist when hemoglobin is below the following levels.[42]

Cut-Off points for the diagnosis of anemia:

	gm/dl (Venous blood)
Adult males	13
Adult females, Non-pregnant	12
Adult females, Pregnant	11
Children, 6 Months to 6 Years	11
Children, 6 Years to 14 Years	12

Different interventions are needed for the prevention and the control of iron deficiency. Iron fortification of staple foods or condiments directed to the whole population is a sustainable and cost-effective approach. However, at some periods of life, especially during pregnancy and in children from the age of 6 months, iron requirements are high. For pregnant women, the current approach favours the daily iron-folate supplementation during pregnancy but the results in terms of public health are disappointing.

For infants and young children, iron fortification of complementary food is effective but it is economically inaccessible to populations with limited resources. When complementary foods are not available, the preventive iron supplementation from 6 to 18 months of age has to be advised. These interventions are more effective when they integrate other approaches like the improvement of the nutritional practice, infection control

and the promotion of breastfeeding and when coupled with programs aiming to control other micronutrient deficiencies. The success of most interventions requires the active participation of the individual.[46]

Information and education of the populations, especially through social mobilization campaigns, are essential because iron deficiency induces few visible symptoms not easily recognizable by individuals. The implementation of national nutrition plans including the control of iron deficiency as one of the priorities and the participation of the public health and the education sectors, food industries, the community and the media should contribute to the success of the intervention and to the control of iron deficiency.

The government of India has initiated a number of national programmes in the country to improve the health and nutritional status of the population.

National Nutritional Anemia Prophylaxis Programme[2]

To control iron deficiency anemia the National Nutritional Anemia Prophylaxis Programme (NNAPP) was launched in 1970 with main intervention of distribution of iron and folic acid tablets to expectant mothers and children.

Antenatal mothers were given 60mg of iron and 500mcg of folic acid for 100days in pregnancy and children were given a tablet 20mg of elemental iron and 100mcg folic acid.

It has been shown that NNAPP did not have the desired impact on anemia prevalence. A multicentric study by Indian Council of Medical Research revealed that 17% of pregnant women had hemoglobin levels less than 9 gm% before iron supplementation was started and compliance rate was unsatisfactory in a considerable proportion of cases. This has been attributed to non-delivery of the tablets to the women, poor quality of tablets and non-consumption of tablets due to lack of awareness about the importance of iron.

Side effects of oral iron therapy are also mentioned as a reason for non-compliance, but these are probably more highlighted than real. It was also noted that as high as 38% of women who had consumed the tablets regularly for more than 90 days during the last trimester had hemoglobin levels less than 10 gm% and in 20% less than 9 gm%. It was felt that 60 mg elemental iron is inadequate. So it was suggested that prophylaxis programme should be converted into a control programme.

National Nutritional Anemia Control Programme

National Nutritional Anemia Control Programme was launched in 1991.

Its objectives are:

1. To assess the baseline prevalence of nutritional anemia in antenatal mothers and young children through estimation of hemoglobin levels. (Now it has been decided that severe anemia should be identified clinically as hemoglobin estimation has limited utility and there is risk of spread of diseases).
2. To put the mothers with more than 10gm% and children with more than 8gm% on the prophylaxis programme.
3. To monitor continuously the quality, distribution and consumption of IFA tablets.
4. To assess periodically the hemoglobin levels of the beneficiaries.
5. To motivate the beneficiary mothers to consume the tablets through relevant nutritional education and pass on the information to their children.

Presently 27 million adults and 30 million child beneficiaries are covered under this programme. Also it has been made an integral part of CSSM programme. And now it is covered under RCH programme.

Pregnant women:

One adult tablet per day for 100 days, each tablet containing 100mg elemental iron and 500mcg of folate and these should be provided after first trimester of pregnancy.

Lactating women and IUD acceptors:

One adult tablet for 100 days.

Preschool children (1-3years):

One small(pediatric) tablet containing 20 mg of elemental iron and 100mg of folic acid for 100 days every year.

Constraints of the present strategy:

The recommended National Nutritional Anemia Control Programme advocating daily hematinics supplementation to pregnant women is not showing the expected results.

These programmes have been ineffective, partly because side effects limit compliance; operational failures are the rule (e.g. Inadequate supplies and poor packaging and presentation of supplements), and health workers and recipients lack appropriate information and motivation[46].

The major constraints are irregular availability of hematinics and lack of education and communication of the importance of hematinics supplementation during pregnancy leading to its poor compliance among subjects.

Alternate strategies

Strategies such as dietary diversification and food fortification have yield significant results in controlling iron deficiency anemia in developed countries. Reducing the prevalence of iron deficiency anemia in developing countries is still a matter of importance. Dietary and food based approaches pose considerable challenges before they can be implemented on a wide scale.

As the programmes did not show effective results in countries such as India, the dosage of iron was increased. Gastrointestinal distress (constipation, nausea, and diarrhea) are often experienced when consuming iron supplements, especially on an empty stomach. Therefore chronic, daily use of iron supplements in excess should be avoided.

There are number of reports associating elevated iron stores with cardiovascular disease and cancer. However, there are no studies that have associated specific chronic intake levels of dietary or supplemental iron with CVD and cancer. It is not possible to estimate how much iron must be consumed to result in a specific level of iron stores.

The upper limit (45mg/day) is for the general healthy population. Individuals with hemochromatosis may not be protected by the upper limit. Relative to an adult, the normal value for hemoglobin is high in neonates, falling to lower-than-adult values by 3-6 months, and rising gradually

thereafter to the adult value by the early teenage years. The role of excess iron in causing intestinal oxidative stress has drawn attention to other approaches of iron supplementation.

Prophylactic administration of iron along with antioxidants like vitamins E and C or foods rich in these vitamins is one such strategy. In order to improve the compliance, reduce the cost of the therapy and reduce the intestinal oxidative stress schedules administering hematinics less than once daily have been tried. Gopalan C[3] and Viteri FE[9] in their studies have indicated that with continuous daily administration, iron absorption could decrease due to "tiredness" of the intestinal mucosa.

According to a study, absorption from a single dose of iron reduces from 30-40% on the first day to as low as 3-6% after a few days of continuous daily administration. Studies carried out on preschoolers support that iron supplementation once or twice a week, increase their hemoglobin status significantly. The potential benefits and shortcomings of these approaches are reviewed.

In experimental animals, the absorption of supplemental iron is greatest when it is administered at times of intestinal mucosal renewal, so that each dose is received by new cell which is based on the mucosal block theory. Thus, inhibition of iron absorption is minimized because of the iron overload in intestinal cells, which occurs with daily iron supplementation.

Because weekly supplementation with iron is effective at improving iron status, this option should be thoroughly explored and evaluated in the context of programs for the prevention and treatment of iron deficiency anemia.

Weekly supplementation programs may improve the logistical and economic constraints that currently limit the provision of supplements to the many target population groups for whom they are recommended, but usually fail to reach. Further work is required to clarify the purpose, delivery and outcomes of iron supplementation programmes.

ADOLESCENT PROGRAMMES IN INDIA

Adolescent girls were not covered under National Nutritional Anaemia Control Programme (NNACP) or any other programme. Interventions for anaemic adolescent girls should raise their iron stores and sustain their hemoglobin at normal levels. This will not only improve their physical and mental capacity, but also subsequently help in reducing the incidence of low birth weight of infants and maternal mortality rates. Recognising the enormous potential of the school system, a project was initiated with support from UNICEF to prevent and control anemia in adolescent girls, utilising the school system.

Adolescent Girls Scheme

The Adolescent Girls Scheme (AGS) or "Kishori Shakti Yojana" is part of the Integrated Child Development Services Scheme (ICDS), devised during 1991-92, for adolescent girls in the age group of 11-18 years.

The scheme fills the gap in services for adolescents including school dropouts, as government schemes previously covered children (0-6 years), mothers and school going children.

The main objectives of the scheme are:

- To improve the nutrition and health status of girls in the age group of 11-18 years;
- To provide literacy and numeracy skills through non-formal education;
- To train and equip adolescent girls to improve or upgrade home-based skills and to enable them to run child care centres at a later stage;
- To promote awareness of health, hygiene, nutrition and family welfare issues and to encourage girls to marry after 18 years.

The scheme provides 'hands-on' learning at the Anganwadi centre, education, health check-ups and supplementary nutrition. A major thrust of the programme is to prevent teenage pregnancies.

UNICEF adolescent anemia project

UNICEF has initiated a project in 11 Indian states to provide iron and folic acid tablets to adolescent girls in order to reduce levels of anemia prior to their initiating childbearing. This strategy has operated through schools with the support of the health centres in some states, and in others, has operated as part of *Kishori Shakti Yojana*, the ICDS adolescent girls' scheme (under the Department of Education and DHFW in Gujarat)**.** Iron and folic acid tablets are provided under supervision of teachers on a weekly basis. Evaluations have shown high rates of compliance and significant improvements in haemoglobin levels (Kotecha et al., 2002). This model of distributing IFA to adolescent girls is now being adopted in the national ICDS programme.

Adolescent Anemia Control Programme

In June 2000, Adolescent Anaemia Control Programme was initiated in Vadodara district, Gujarat, covering over 69000 school girls. The main objective of the programme was to improve the poor iron status of the adolescent girls. The intervention primarily consisted of weekly IFA supplementation to school girls under direct supervision of class teacher and nutrition education to the beneficiary girls and teachers with the help of Information Education and Communication (IEC) material. Each IFA tablet contained 100 mg elemental iron and 0.5 mg folic acid.

Many studies of weekly iron supplementation showed improvement in increase in hemoglobin and iron stores. Added Vitamin C to the existing weekly iron/folate regiman had some advantages in absorbtion and increase in hemoglobin, this should be thoroughly explored and evaluated in the context of programs for the prevention and treatment of iron deficiency anemia.

LITERATURE REVIEW

Iron deficiency anaemia is the most prevalent micronutrient deficiency among humans all over the world. Adolescent period is the formative period of life when the maximum amount of physical and psychological and behavioral changes takes place and they are a particularly vulnerable group as their requirements of iron, as well as its losses from the body are high. Anaemia during adolescence limits growth and delays the onset of menarche, which in turn may lead to cephalopelvic disproportion. Very often, in India, girls get married and pregnant even before the growth period is over, making anaemia doubly risky. Since the anaemic status of the adolescent girls is bound to affect their offspring, care during this period is likely to pay rich dividends.

An intermittent supplementation of iron, that is, weekly once or twice could be effective, costs less, has fewer side-effects and would be of particular use in a public health programme, if proved to be effective.

Studies regarding comparing Daily and weekly iron supplementation

Kianfar H et al (2000)[31] in their study conducted in high school girls have concluded a weekly iron dose was as effective as a daily dose in treating anemia but the daily dose was more effective in improving iron stores than a weekly dose in the short run.

The study conducted by **Young MW(2000)**[32] in pregnant women of Northern Malawi has proved that a weekly iron supplement has similar haematologic effects, and less side effect profile, in comparison with a standard daily supplement when administered through an existing primary healthcare programme, although both regimens are relatively unsuccessful in the reduction of anemia prevalence during pregnancy.

Zavaleta et al (2000)[34] have concluded in their study that daily supplements led to higher Hb increases than intermittent supplements. Thus, both iron supplementation schedules were efficacious in preventing iron deficiency in adolescent girls through the school system, and the daily schedule was better than the intermittent schedule at increasing Hb values and reducing anemia.

A Chinese study done by **Gillespie S (1998)**[24] has shown that weekly iron supplementation with 120 mg was more effective than daily 60mg dose.

Berger et al (1997)[30] in their study have said weekly iron supplementation is as effective as 5day per week iron supplementation in Bolivian school children living at high altitude.

Gillespie S (1998)[24] in his study has shown that weekly iron supplementation with 120mg was more effective than daily 60mg dose which in turn was as effective as 120 mg dose. Another study done by the same author revealed that weekly 180mg iron was less effective than daily

60mg dose. While iron supplements are needed in certain groups and in particular regions, increases dietary intakes could be supplied by food fortification as well as by individual improvements in intake. A proposed meta-analysis is required to finally come to a conclusion about the degree of effectiveness of intermittent dosage.

Study done by **Gilgen G et al (2001)**[33] showed that the rate of increase in haemoglobin levels and the correction of anemia is faster in the daily iron supplementation group when compared with other groups. Weekly iron is equally effective in combating anemia. But the supplementation has to be continued on a longer basis in order to combat both anemia and iron deficiency.

Soemantri AG (1997)[29] et al in their study conducted in 144 children have concluded that the hemoglobin levels in both daily group and weekly group increased significantly.

Berger J (1997)[30] in his study said final hemoglobin and its changes were similar in both supplemental groups.

Agarwal K N, Gomber S, Bisht H, Som M[35] study showed the daily intake for 100 days raised hemoglobin level, which was maintained until four months after withdrawal of intervention. Regular weekly administration was effective and seems suitable for populations with mild to moderate anemia.

Studies regarding Weekly iron supplementation

Beasley et al (2000)[28] in their study have said that weekly iron supplementation may be effective means of increasing iron stores.

P.V. Kotecha, S. Nirupam & P.D. Karkar[41] study concluded that supervised, once a week IFA supplementation to adolescent girls through institutions specially schools was found to be an effective intervention to reduce anaemia and was scalable within the system.

Studies regarding iron supplementation along with other micronutrient supplementation

Persson V et al (2000)[25] have said programmes to improve iron status should consider including both vitamin A prevention programmes and deworming.

Kolsteren P et al (1999)[26] in their study on women with anemia have showed that addition of vitamin A and zinc to the treatment for anemia can increase hemoglobin levels more than with iron alone.

Ahluwalia (2002)[29]**, Madhavan Nair (2001)** in their studies have stressed on the aspect of improved intake in the correction of anemia.

Studies regarding iron supplementation along with Vitamin C supplementation

Palupi L, Schultink W, Achadi E, Gross R[10] showed that the dietary intake of Vitamin C along with iron rich sources enhances absorption of the iron significantly[10].

A study done by **Davidsson L**[37], it was observed that supplementation of 50mg of vitamin C along with iron folate increased the iron absorption to 7.7% as compared to iron supplementation alone where the iron absorption was 1.6% only.

Pande Rohini, Kuz Kathleen, Wodia Sunayana, Mac Quarrie Kerry, Jain Saranga.[50] showed supplementation of iron would be effective if Vitamin C rich foods consumed along with iron supplementation.

A study done by **S Mehnaz, S.Afzal, S.Khalil, Z.Khan**[36] showed the difference in the response of mean increase in Hb% of the subjects getting Vitamin C supplementation with Fe/folate as compared to those receiving only iron/ folate.

A Study done by **Sharma A, Prasad K, Rao KV**[20] on adolescent girls of poor community it was observed that weekly supplementation of vitamin C along with iron/folate had a significant higher increase in haemoglobin concentration (0.76g/dl), as compared to weekly iron/folate supplementation alone (0.71 g/dl) after three months of supplementation[20].

STUDY JUSTIFICATION

In India Iron deficiency anemia prevalence depending on the age and sex, reported to range from 38-72 percent, majority of them being women and children. The iron deficiency anemia prevalence rate beyond the age of six years is higher in girls. This could be due to certain factors such as food habits, gender discrimination in food allocation, menstruation and early marriage leading to early pregnancy. Estimates suggest that 25-50% girls become anemic by the time they reach menarche. Thus in these adolescent girls pregnancy only serves to aggravate their pre-existing anemia.

Iron deficiency anemia is prevalent throughout the world because of the inefficient absorption of nonheme iron, which forms the bulk of the iron in diet. Absorption of this type impaired by substances in the food like phytates which reduces its bioavailability. Ascorbic acid reverses the effect of dietary inhibitors and is one of the most powerful known promoters of nonheme iron absorption. Though supplementation of iron and folic acid remains the cornerstone of the treatment and prevention of anemia, addition of vitamin C has its other added advantages.

We continued to have various health programmes to prevent anemia among young children and adolescents for the past several years but we continued to have higher prevalence of anemia among them which leads to morbidity and mortality during pregnancy and childbirth and after delivery.

The major reason for continuation of higher rate of anemia is the ineffective regular administration of iron and the daily drug regimen has got some adverse effects which hinders continuous intake of iron and also giving iron daily for large number of children and adolescents appears to be practically not feasible. Indian diets are cereal based and contain high phytate content which reduces the bioavailability of iron which is significantly improved by added vitamin C in the weekly supplementation and also easy to administer.

This study has been done in view of the efficacy of added vitamin C in combating the anemia in adolescent girls to the weekly iron/folate regimen.

AIM OF THE STUDY

To compare the efficacy of the weekly oral iron/folate versus weekly oral iron/folate with Vitamin C supplementation in anemic adolescent school girls aged 10 to 14 years

MATERIALS AND METHODS

This is a prospective study. Hypothesis of the study is comparing the efficacy of supplementation of weekly iron tablets with and without vitamin C which will improve the Hb% in anemic adolescent girls. Based on this hypothesis equivalent study design was selected.

Four government corporation schools located at Begam Sahib street, Triplicane high road, Veera Perumal kovil street, Vannia Teynampet Eldams Road affiliated to Government Royappettah Hospital and Kilpauk Medical College Hospital selected for this study. Adolescent girls in the age group of 10 to 14 years studying 6^{th} to 9^{th} standard were selected for this study. Permission from school authorities was obtained. Study was started in August 2009 and was conducted till April 2010.

EXCLUSION CRITERIA:

1. Children known to have hemolytic anemia, bleeding disorder and leukemia.
2. Known anemic child taking treatment outside.
3. Children with chronic illness.
4. Those who are not willing to participate in the trial.

All these children were excluded from this study. For alpha error 0.05 and beta error of 0.2 with 95% confidence interval for the outcome i.e., increase in Hb% about 95 children are needed in each group.

Permission from school authorities were obtained. Ethical committee approval from the college was obtained. Informed parental consent was obtained from each of the children. 20 microlitre of blood was obtained from the children who has given consent (322) by finger prick method and sample is estimated for Hb% using cyanmethemoglobin method by adding 5ml drabkins solution and results were read by spectrophotometer[12.]

Two children from Triplicane high road and 2 child from Eldams road school were found to be severely anemic (Hb%<7gm/dl) and referred to tertiary care hospital for further management. Children with Hb% >12gm/dl (98 children) were excluded from the study. 220 children with mild and moderate anemia (Hb% 7 to 12 gm/dl) were totally enrolled for this study and after randomization 2 groups were divided with each containing 110 children.

Interventions:

Group A: 110 children receiving weekly oral iron (100mg of elemental iron 500mcg of folic acid) tablet for 24 consecutive weeks.

Group B: 110 children receiving weekly iron tablet (100mg of elemental iron 500mcg of folic acid) with Vitamin C 100mg tablet for 24 consecutive weeks.

After forming two groups using randomization baseline data of Hb%, height and weight were recorded. Anthropometry measurements like weight in kg with sensitivity of 100gm, Height in cm using non stretchable tape with

sensitivity of 1cm were recorded. Body mass index was calculated using NCHS standards (CDC chart) for each of these anemic children. Qualitative data was obtained through self-administered questionnaire regarding symptoms of anemia from the parents after counseling before the study. The symptoms were given a score of 0-Not satisfactory, 1-Average, 2-Good. Awareness questions regarding anemia were asked through self-administered questionnaire.

All adolescent girls participating in the study were initially dewormed with the tablet Albendazole 400mg and then started on oral supplementation.

The iron tablets(contains 100mg of elemental iron and 500microgram of folic acid) and Vitamin C(100mg) were supplied by the Government. Tablets were given to the enrolled girls on every Monday of the week by the school teacher in the afternoon half to 1hour after lunch. They were advised to take tablets along with water, only after food and never in the empty stomach and under supervision.

Parents were asked to report to the principal investigator through the class teacher, if their children developed any problem during the study period. Contact phone number of the principal investigator was given to the class teacher.

Any side effects of the drugs like abdominal pain, vomiting, and loose stool were documented as reported by the children or their parents to the teacher and later confirmed by the investigator.

At the end of 12 weeks blood sample was taken for Hb% and measured by cyanmethemoglobin method and height and weight were recorded. The same method was repeated after 24 weeks. Qualitative data was also obtained through self-administered questionnaire regarding anemia. Data of 105 children from each group who completed the study were available for statistical analysis. Results were tabulated and analysed.

ANALYSIS OF OBSERVATIONS

In this study, 346 adolescent girls in the age group of 10-14 years studying in corporation schools were taken, of this 322 had given consent for screening, and baseline data of height, weight, hemoglobin status obtained. Of this 322 subjects, 98(30.43%) had Hb levels >12gm/dl, they were excluded from the study. 4 severely anemic girls (<7gm/dl) were referred to tertiary care hospital for further management. 220 girls with Hb% <12gm/dl were participated in this study they were randomly divided into two groups comprising 110 in each group, their baseline data of hemoglobin, height and weight were recorded.

The girls in Group A were supplemented with iron folic acid tablet(100mg elemental iron and 500mcg of folic acid) weekly, and in Group B were supplemented with Iron folic acid(100mg elemental iron and 500mcg of folic acid) with Vitamin C 100mg weekly for 24 consecutive weeks. Finally 105 children in Group A, 105 children in Group B were available for statistical analysis. 5 children in each group lost their follow up and dropped from the study.

Data were analysed as follows:

a) Prevalence of anemia

b) Age distribution

c) Nutritional status

d) Awareness about factors regarding anemia

e) Height at enrollment

f) Weight at enrollment

g) Hemoglobin estimation at enrollment

h) Severity of anemia

i) Mean hemoglobin distribution

j) Compliance

k) Side effects

l) Outcome analysis

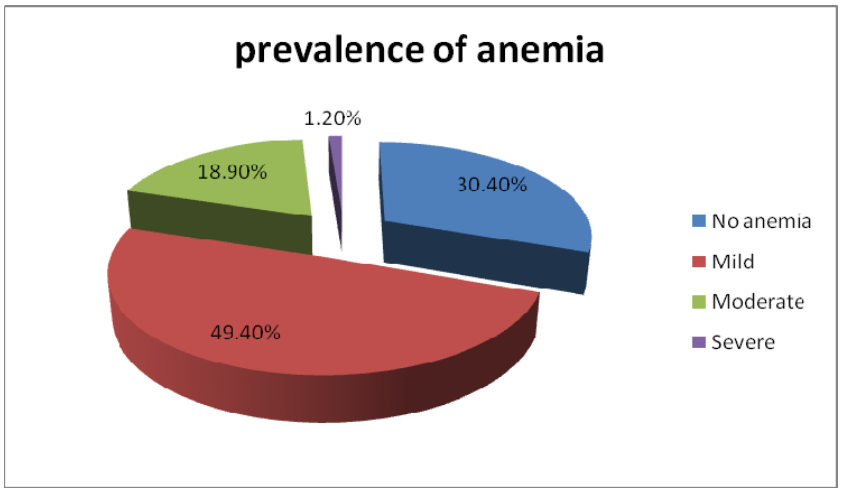
m) Analysis of associated factors influencing the outcome.

n) Quality data analysis

A) Prevalence of anemia

Prevalence of anemia of the study population is 69.6% and in that 1.2% is severely anemic

Prevalence of Anemia at enrollment

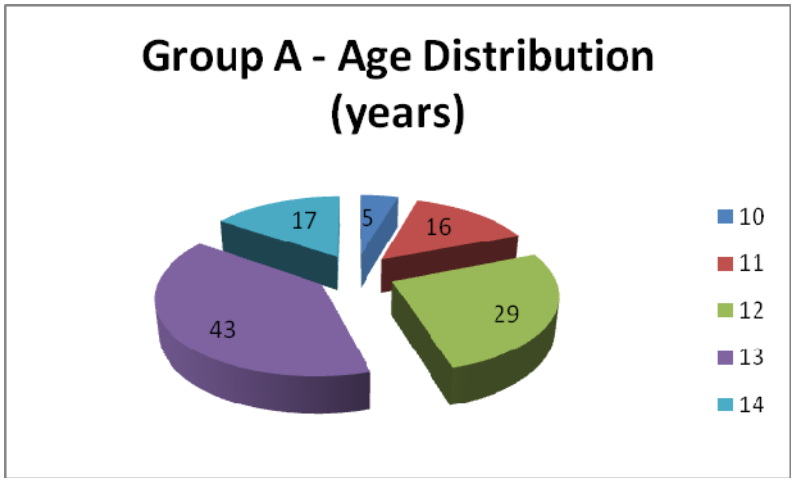


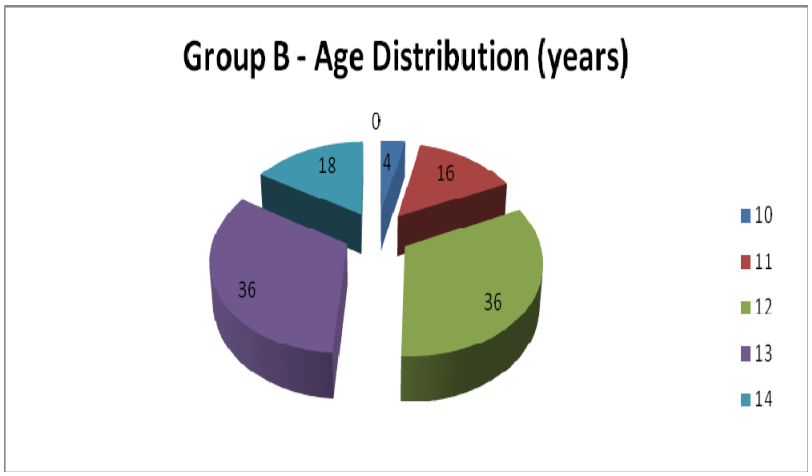
B) Age distribution

Table–1: Age distribution

AGE	Group A	Group B
10	5(4.5%)	4(3.6%)
11	16(14.6%)	16(14.6%)
12	29(26.4%)	36(32.7%)
13	43(39.0%)	36(32.7%)
14	17(15.5%)	18(16.4%)
Total	110(100%)	110(100%)

Majority of the children were in the age group of 12 and 13 years. The age distribution was comparable between two groups.





C) **Nutritional status**:

Table-2: Nutritional Status

Nutritional Status	Group A	Group B
Normal	79(71.8%)	78(70.9%)
Undernutrition	27(24.5%)	25(22.7%)
Overweight	4(3.6%)	7(6.4%)

Using body mass index (NCHS standard-CDC chart)

Normal nutritional status were seen in 71.8%(79) children in Group A and 70.9%(78) of children in Group B. Undernutrition were seen in 24.5% (27) of children in Group A and 22.7%(25) of children in Group B. Overweight were seen in 3.6% (4) of children in Group A and 6.4%(7) of children in Group B. Most of the children were in the normal nutritional status range. Both groups were comparable in their nutritional status.

D) Awareness about Anemia

Awareness questionnaire was undertaken related to causative factors, impact, prevention of anemia on an average only 7.3% in Group A and 9.4% in Group B were aware about the factors related to anemia.

Table -4: Awareness Factors about anemia

S.No	Factors regarding anemia	Group A (Aware)	Group B (Aware)	N=
1	Tea/coffee intake	8(7.3%)	7(6.4%)	110/110
2	Worm infestation	10(9.1%)	12(10.9%)	110/110
3	Cognitive factors	8(7.3%)	12(10.9%)	110/110
4	Delivery related complications	6(5.5%)	11(10%)	110/110
5	Iron rich foods	9(8.2%)	10(9.1%)	110/110
6	Affects adolescent girls	7(6.4%)	10(9.1%)	110/110

E) Height at enrollment

Table -5 :Height at enrollment

	Mean	**SD**
Group A	142.8	8.3
Group B	144.2	8.3

The mean height of girls in Group A were 142.8cm and standard deviation 8.3cm, in Group B were 144.2cm, standard deviation 8.3cm and were comparable.

F) Weight at enrollment

Table-6:Weight at enrollment

	Mean	**SD**
Group A	33.4	8.6
Group B	33.3	7.8

Mean weight in Group A were 33.4kg, standard deviation 8.6kg and in Group B were 33.3kg, standard deviation 7.8kg and were comparable in both groups.

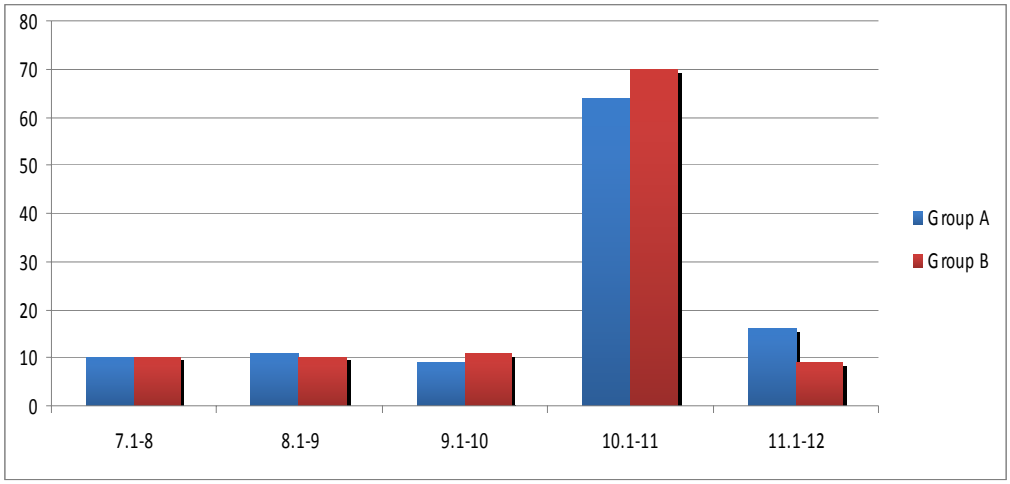
G) Hemoglobin Estimation at enrollment

Table-6: Hemoglobin Distribution

Hb%(gm/dl)	Group A	Group B
7.1-8.0	10(9.1%)	10(9.1%)
8.1-9.0	11(10%)	10(9.1%)
9.1-10.0	9(8.2%)	11(10%)
10.1-11.0	64(58.2%)	70(63.6%)
11.1-12.0	16(14.5%)	9(8.2%)
Total	110(100%)	110(100%)

No of children with Hb% in Group A and Group B in Hb% between 7.1 to 8 gm/dl were 10(9.1%), 10 (9.1%) respectively. Children with Hb% between 8.1 to 9 gm/dl in Group A and Group B were 11(10%), 10(9.1%) respectively. Hb% between 9.1 to 10 gm/dl in Group A and Group B were 9 (8.2%), 11(10%) respectively. Hb% between 10.1 to 11 gm/dl in Group A and Group B were 64(58.2%), 70(63.6%) respectively. Hb% between 11.1 to 12 gm/dl in Group A and Group B were 16(14.5%), 9(8.2%) respectively.

Hemoglobin Distribution at Enrollment



H) Severity of Anemia

Table-7: Severity of anemia

Grades	Group A	Group B
Moderate (7 to 9.9 gm/dl)	30(37.5%)	31(39.4%)
Mild (10 to 11.9 gm/dl)	80(62.5%)	79(60.6%)

According WHO criteria for severity of anemia, moderate anemia was seen in 37.5% in Group A, 39.4% in Group B, mild anemia was seen in 62.5% in Group A, 60.6% in Group B. Both groups were comparable in terms of severity of anemia at enrollment.

I) MEAN Hb% DISTRIBUTION

Table-8: Mean Hb% distribution

	Group A		Group B		P value
	Mean	SD	Mean	SD	
Hb% (gm/dl)	10.29	1.14	10.26	1.08	0.86 Not significant

Mean Hb% distribution in Group A is 10.29gm/dl, and standard deviation is 1.14gm/dl, and in Group B is 10.26gm/dl, and standard deviation is 1.08gm/dl. Independent t test showed the p value 0.86 which is statistically not significant. Both groups were comparable.

J) Compliance

About 90% of children did not skip even single tablet in both groups, 5 children in each group lost their follow up due to long absent and transferred to other schools.

K) Side effects

Table-9: Side effects

Side effects	Group A	Group B
Vomiting	7	6
Pain abdomen	4	4
Loose stool	0	1

Vomiting occurred in 7 children in Group A and 6 children in Group B. 4 children in each group had abdominal pain. 1 child in group B had loose stools. But all these side effects were only mild and these did not interrupt from taking treatment. Side effects were comparable between groups.

L) OUTCOME ANALYSIS

Table-10: Increase in Hb% - Group A (WEEKLY IFA ONLY)

Group A	Initial		At 12 weeks		At 24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Hb% gm/dl	10.29	1.14	11.18	1.07	11.80	0.93

Mean hemoglobin at the beginning of the study period was 10.2gm/dl and its standard deviation was 1.14gm/dl. Mean hemoglobin at the end of 12 weeks period in Group A was 11.18 and standard deviation was 1.07. Mean hemoglobin at the end of 24 weeks was 11.8 and standard deviation was 0.93.

Table-11: Increase in Hb%- Group B (WEEKLY IFA+VITAMIN C)

Group B	Initial		At 12 weeks		At 24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Hb% (gm/dl)	10.26	1.08	11.72	0.89	12.54	0.71

Mean hemoglobin at the beginning of the study period in Group B was10.26gm/dl and its standard deviation was 1.08gm/dl. Mean hemoglobin at the end of 12 weeks period in Group B was 11.72 and standard deviation was 0.89. Mean hemoglobin at the end of 24 weeks was 12.54 and standard deviation was 0.71.

Table-13: Comparision of Hb% between Group A and Group B at 12 weeks

	Group A		**Group B**		**P value (T test)**
	Mean	**SD**	**Mean**	**SD**	
Hb%(gm/dl)	11.18	1.07	11.72	0.89	0.000(significant)

At 12 weeks there is a statistically significant (p=0.000 i.e., p<0.05) increase in Hb% in Group B compared to Group A.

Table-14: Comparison of Hb% between Group A and Group B at 24 weeks

	Group A		**Group B**		**P value (T test)**
	Mean	**SD**	**Mean**	**SD**	
Hb% (gm/dl)	11.8	0.93	12.54	0.71	0.000(p<0.05) significant

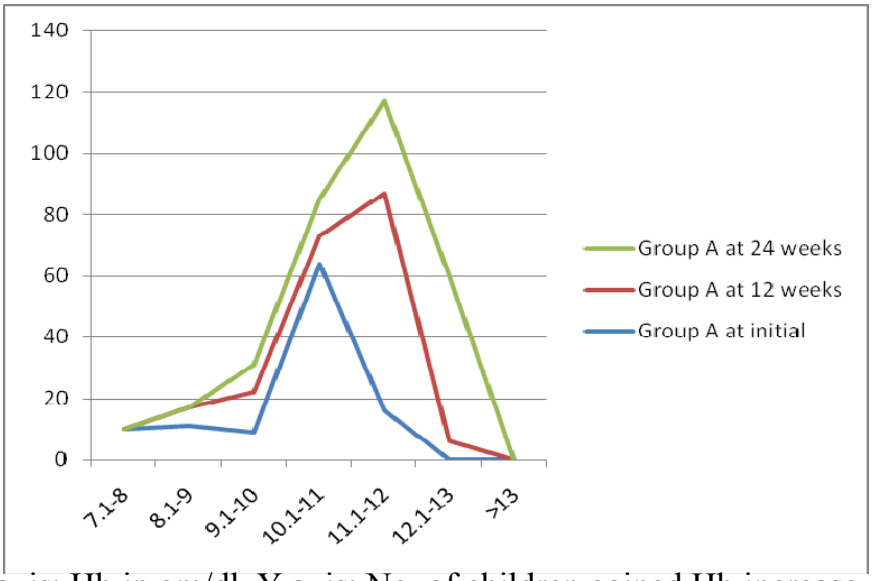
At 24 weeks also there is statistically significant (p=0.000) increase in Hb% in Group B compared to Group A.

Table-15: Hemoglobin Gain

	Study Period	**Group A**		**Group B**		**P value (T test)**
		Mean	**SD**	**Mean**	**SD**	
Hb% (gm/dl)	0- 12wks	0.92	0.35	1.47	0.57	P=0.000 (significant)
	12-24 wks	0.63	0.35	0.82	0.39	P=0.000 (significant)
	0-24 wks	1.56	0.47	2.30	0.69	P=0.000 (significant)

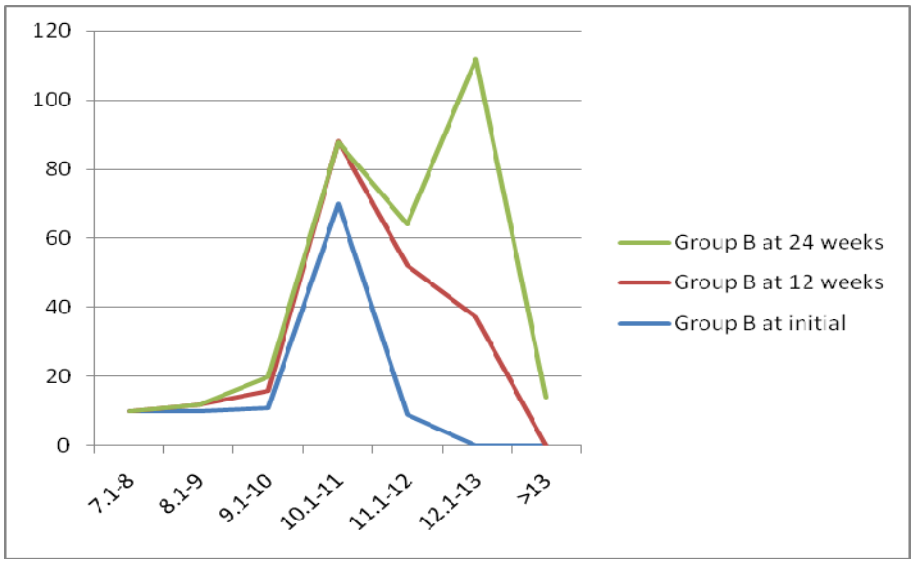
There is mean gain in hemoglobin level in Group A at 12 & 24 weeks were 0.92±0.35, 1.56±0.47, in Group B 1.47±0.57, 2.30±0.69. In between 12 to 24 weeks 0.63±0.35, 0.82±0.39. But in Group B there is statistically significant increase in Hb% gain i.e., p=0.000 ($p<0.05$)) in the initial 12 weeks period and also at the end of 24 weeks and also in between 12 to 24 weeks compare to Group A.

Group A-Hemoglobin distribution before & after intervention



X axis: Hb in gm/dl Y axis: No. of children gained Hb increase

Group B – Hemoglobin distribution before & after intervention



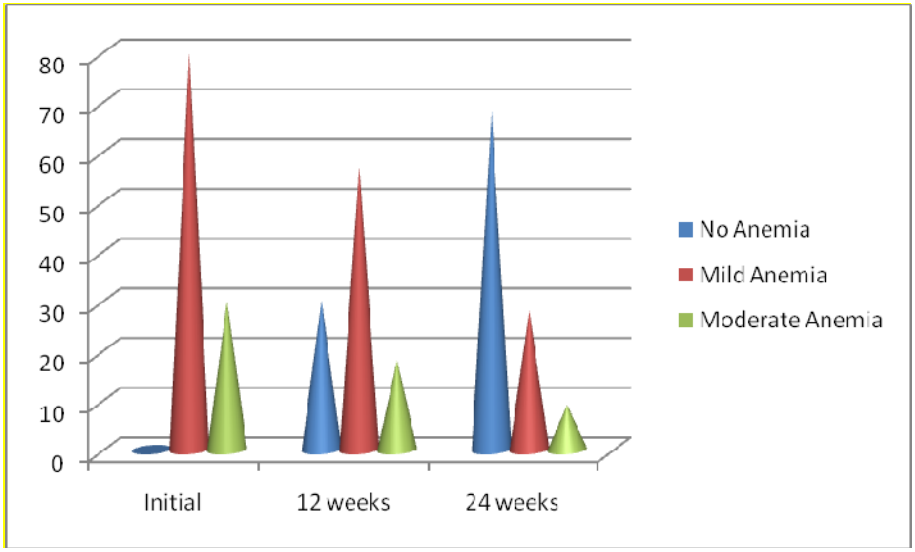
X axis: Hb in gm/dl Y axis: No. of children gained Hb increase

SEVERITY OF ANEMIA – GROUP A

Table-16: Group A – Severity of anemia

Grades of Anemia	Initial	12 weeks	24 weeks
Moderate	30(37.5%)	18(17.1%)	9(8.6%)
Mild	80(62.5%)	57(54.3%)	28(26.7%)
Normal	0(0%)	30(28.6%)	68(64.8%)

The severity of anemia in Group A decreased to 17.1% from 37.5% in moderate, and to 54.3% from 64.5% in mild variety at 12 weeks, and at 24 weeks it becomes 8.6% in moderate anemia, 26.7% in mild anemia.

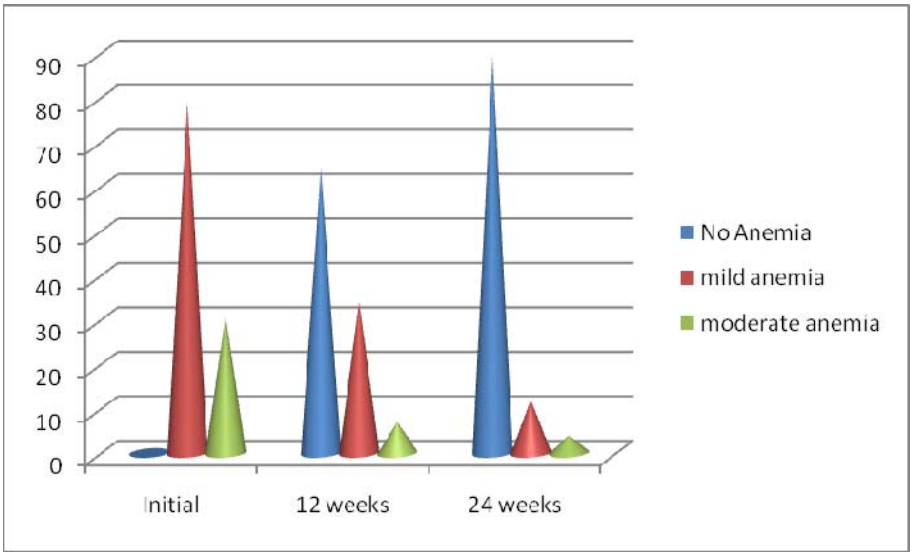


SEVERITY OF ANEMIA –GROUP B

Table-17: Group B-Severity of anemia

Grades of Anemia	Initial	12 weeks	24 weeks
Moderate	31(39.4%)	7(6.7%)	4(3.8%)
Mild	79(60.6%)	34(32.4%)	12(11.4%)
Normal	0(0%)	64(61.0%)	89(84.8%)

In Group B severity of anemia decreased from 39.4% to 6.7% in moderate anemia, 60.6% to 32.4% in mild anemia at the end of 12 weeks, and to become 3.8%in moderate and 11.4% in mild anemia at the end of 24 weeks.



No. of Normal children after 24 weeks of supplementation

Table-18: Group A-children with normal Hb% at 24 week (WEEKLY IFAONLY)

Period	**Initial**	**At 24 weeks**			
Grade of anemia	**N=110**	**Moderate anemia**	**Mild anemia**	**No anemia**	N=105
Moderate	30(37.5%)	9	17	3(2.9)%	
Mild	80(62.5%)	-	10	66(62.9%)	

In group A at the end of 24 weeks 3(2.9%) & 66(62.9%) girls become not anemic from moderate and mild anemia respectively from 30 & 80 at enrollment.

Table-19: Group B-Not anemic at 24 weeks (WEEKLY IFA+VIT.C)

Period	**Initial**	**At 24 weeks**			
Grade of anemia	**N=110**	**Moderate anemia**	**Mild anemia**	**No anemia**	N=105
Moderate	31(39.4%)	4	8	18(17.1%)	
Mild	79(60.6%)	-	-	75(71.4%)	

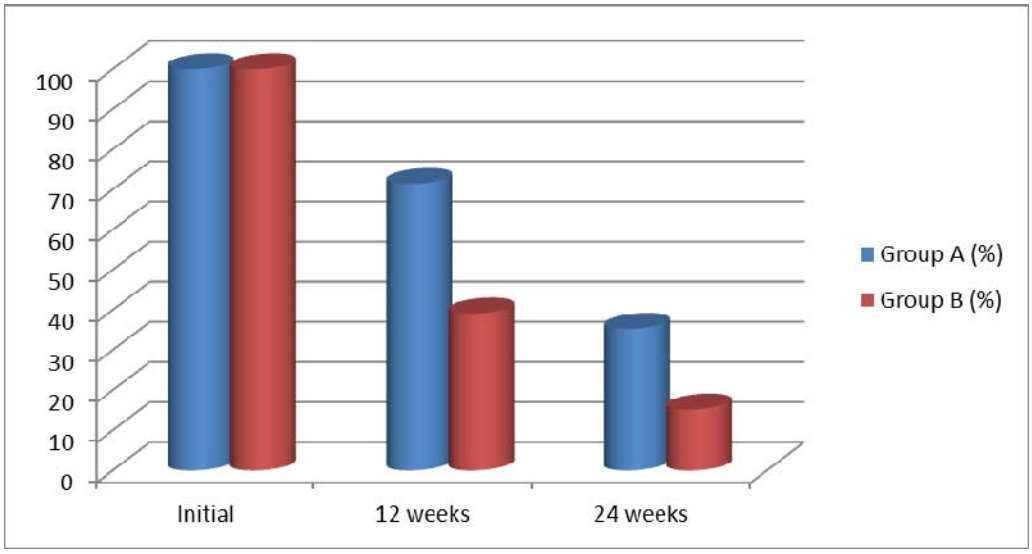
In group B at the end of 24 weeks 18(17.1%) & 75(71.4%) girls become not anemic from moderate and mild anemia respectively from 31(39.4%) & 79(60.6%) at enrollment. It shows that conversion into non anemic status from low hemoglobin is much better in children taking Fe/Folate with Vitamin C.

PREVALENCE OF ANEMIA

Table-20: Prevalence of Anemia by Periods of standard

Period of study	Group A	Group B	N=
Initial	100%	100%	110
12 weeks	75(71.4%)	41(39.1%)	105
24 weeks	37(35.3%)	16(15.2%)	105

Prevalence is decreased at the end of study to 37% in Group A, and 15.2% in Group B.



Height

Table-21: Height increase

	Initial		At 12 weeks		At 24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Group A	142.8	8.3	144.1	8.2	144.9	8.1
Group B	144.2	8.3	145.6	8.2	146.3	8.3
P value	0.23		0.73		0.20	

There is no statistically significant difference in mean height increase between two groups at 12 weeks and 24 weeks of the study period.

Height Gain

Table-22: Height Gain

	0-12weeks		12-24 weeks		0-24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Group A	1.3	0.5	0.8	0.3	2.1	0.8
Group B	1.5	0.5	0.7	0.4	2.2	0.5
P value	0.04(significant)		0.09		0.47	

There is a statistically significant increase in height gain in Group B compared to Group A at the end of 12 weeks period only but at the end no significant difference.

Weight

Table-23: Weight increase

	Initial		At 12 weeks		At 24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Group A	33.4	8.6	34.7	8.5	35.4	8.5
Group B	33.3	7.8	34.5	8.0	35.4	8.0
P value	0.91		0.91		0.97	

There is no statistically significant increase in mean weight between two groups at 12 weeks and at 24 weeks of the study period.

Weight Gain

Table-24: Weight Gain

	0-12 weeks		12-24 weeks		0-24 weeks	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Group A	1.3	0.6	0.7	0.4	2.0	0.7
Group B	1.3	0.5	0.8	0.4	2.0	0.6
P value	0.90		0.06		0.34	

There is no statistically significant increase in weight gain in between groups during the study period.

M) Analysis of associated factors influencing the outcome.

Table – 25

	Group A		Group B		T test
	Mean	**SD**	**Mean**	**SD**	
Weight	35.39	8.53	35.35	8.01	p = 0.97 (not significant)
Height	144.9	8.06	146.3	8.25	p = 0.20 (not significant)
BMI	17.08	3.01	17.35	2.8	p = 0.84 (not significant)

There was no statistically significant difference in weight, height, BMI gain between the two groups.

N) Quality Data Analysis

Table - 26

S.NO	DATA		0	1	2	McNemar Bowker test
1	Memory	Initial(%)	10	83.3	6.7	0.000
		Final	1	82.3	16.7	
2	Activity	Initial	9.5	82.9	7.6	0.000
		Final	1	78.6	20.5	
3	Games	Initial	9.5	82.9	7.6	0.000
		Final	1	78.6	20.4	
4	Studies	Initial	10	83.3	6.7	0.000
		Final	1	82.3	16.7	
5	Apetite	Initial	11	81.9	7.1	0.000
		Final	0	53.8	46.2	
6	Sleep	Initial	11	81.9	7.1	0.000
		Final	0	56.2	43.8	
7	Cough	Initial	6.7	4.3	89.0	0.001
		Final	5	3.3	96.2	
8	Overall	Initial	10.5	82.4	7.1	0.000
		Final	0	57.6	42.4	

p value < 0.05 (significant)

In both groups memory, activity, appetite, sleep pattern, reduction of cough symptom significantly improved after supplementation of tablets.

SUMMARY

- We followed 220 anemic adolescent school girls aged 10-14 years with Hb% level of 7-12gm/dl and were divided into two groups.
- 110 girls in Group A with weekly once Iron/Folate and 110 girls in Group B with weekly Iron/Folate with Vitamin C 100mg were supplemented for 24 consecutive weeks. With the baseline Hb%, height and weight it was again recorded at 12 and 24 weeks results were analysed for 105 children in each group.
- Awareness questionnaire before the study and quality data before and after the study were taken.
- Study revealed that mean hemoglobin increase noticed in both groups but there is statistically significant increase in mean hemoglobin noticed in the girls who had supplemented with weekly once iron folic acid with Vitamin C. Also the rate of rise in hemoglobin and decrease in severity and prevalence of anemia were significant in that group.
- There is no difference in weight and height gain and BMI in both groups. Awareness about anemia was poor and the quality data showed better response in both groups and this is subjective improvement, it may be influenced by various other factors also.

DISCUSSION

Iron deficiency anemia is the significant problem in adolescent girls studying in the schools. A study on adolescents by Mehtha[52] revealed that 63.8% girls were anemic[39], another study by Shaw NS[40] showed iron deficiency was more prevalent in teenage girls, and another study by Cai MQ, Yan WY[39] showed 61.8% anemia prevalence in adolescent girls. Our study showed about 68.3% of young adolescent girls were anemic and the prevalence is comparable. In that49.4% of the girls were mildly anemic, 18.9% were moderate and 1.2% were severely anemic.

In order to improve the compliance and reduce the intestinal oxidative stress, schedules administering hematinics less than once daily have been tried. This strategy is based on intestinal mucosal turnover time. It has been suggested that saturation of iron binding protein, apoferritin, in the mucosal cells is the rate limiting step of the absorption of iron (mucosal block theory)[35]. Since the mucosal removal time in humans is 5-6 days, the strategy of weekly iron supplementation has been strongly suggested by some worker [11,13] and it also has been done in our study.

Compliance is one of the important problem in iron supplementation, it is better and side effects are less in twice weekly supplementation schedule done by Shoba S and Sharda D in adolescent school girls in India[44]. In our

study both groups showed better compliance and no major side effects were noticed.

Many studies revealed that weekly iron supplementation in adolescent girls had a reasonable increase in hemoglobin concentration.

Mehta et al.[52] reported an increase in the mean haemoglobin level from 10.45±1.21 g/dl to 11.99±1.19 g/dl and the halving of the prevalence of anaemia from 63.8% after weekly supplementations of iron and folic acid for 25 weeks, which were comparable with daily supplementation. A study done by Cai MQ, Yan WY[39] showed that weekly supplementation of iron improved hemoglobin concentration in adolescent girls. A study done by Kotecha et al.[41] showed that weekly iron supplementation in adolescent girls had improvement in both hemoglobin and serum ferritin levels. In our study weekly once IFA supplementation showed comparable results with a mean Hb% increase from 10.29±1.14 to 11.8±0.93 g/dl at the end of 24 weeks.

The dietary intake of Vitamin C along with iron rich sources enhances absorption of the iron significantly[22, 23]. In a study done in Tamilnadu by Swarnalatha A, Yegammai C concluded that adolescent girls showed better absorption of iron if it is given with absorption enhancers like Vitamins A and C than iron alone[19]. In a study by Davidsson L.[37] it was observed that supplementation of 50mg of vitamin C along with iron folate increased the iron absorption to 7.7% as compared to iron supplementation alone where

the iron absorption was 1.6% only. In our study 100mg Vitamin C is added to the weekly regimen and showed comparable results.

A study done by S Mehnaz, S.Afzal, S.Khalil, Z.Khan[36] showed the difference in the mean increase in Hb% response of the subjects getting Vitamin C supplementation with Fe/folate as compared to those receiving only iron/ folate.

A study by Sharma et al.,[20] done on adolescent girls of poor community it was observed that weekly supplementation of vitamin C along with iron/folate had a significant higher increase in haemoglobin concentration (0.76g/dl), as compared to weekly iron/folate supplementation alone (0.71 g/dl) after three months of supplementation.

In our study the weekly iron supplementation with added Vitamin C showed that increase in hemoglobin concentration of 2.30±0.69g/dl compared to a hemoglobin increase of 1.56±0.47g/dl in weekly once iron folate supplementation group at the end of 24 weeks period and it was significant, and it also showed at the end of 12 weeks also there is a significant increase in concentration of hemoglobin(0.82±0.39g/dl) in the weekly IFA+Vitamin C supplementation group. The prevalence and severity is also significantly reduced in the added Vitamin C group.

In our study rate of rise of hemoglobin was more in the girls who had lower hemoglobin level before supplementation in the weekly IFA with

Vitamin C group as showed in the study done by S Mehnaz, S.Afzal, S.Khalil, Z.Khan[36].

Weekly supplementation is superior to daily supplementation on height gain among school children in Thailand done by Rassamee Sungthong, Ladda Mo-suwan, Virasakdi Chongsuvivatwong and Alan F Geater[45]. In our study there is height gain occurred in both groups, slight marginal increase occurred in Fe/folate with Vitamin C group in the 12 weeks period but no significant height gain difference noticed between the groups at end of the study.

In the study done by Shubadha J. Kanani and Rashmi H. Poojara[38] has showed significant weight gain noticed in the adolescent school girls of 10-14 years. In our study also weight gain noticed in both groups in 12 weeks and 24 weeks period but there is no significant difference in weight gain noticed between the groups.

Study on the impact of iron and folic acid supplementation along with Vitamin C on haemoglobin Status of adolescent girls in an ICDS block in 2008 report showed when IFA supplementation was given with vitamin C bi-weekly to the subjects compared to once weekly and daily iron with and without vitamin C, the increment of hemoglobin observed was maximum. The increment in mean height was 1.0 cm at three months and 0.2 cm at six months of supplementation. The mean weight of the subjects increased by

2.2 kg at three months and it remained the same at six months of supplementation. The haemoglobin increased by 2.5 per cent at three months and 0.2 percent at six months of supplementation. In our study we used weekly once supplementation it showed comparatively better results.

From the Indian Institute of Health and Family Welfare, Annual Report, 2001-2002[51] it was evident that the results of midterm survey revealed that weekly IFA supplementation to school going girls, under teacher's supervision, for preceding six months together with IEC intervention resulted in a significant increase in haemoglobin levels, indicating the feasibility of this approach. In our study also showed that added Vitamin C to the weekly IFA regimen under supervision is feasible in improving the hemoglobin level significantly.

Qualitative analysis showed that significant improvement in memory, physical activity, appetite etc., in both the groups after intervention. But this is only subjective and several other factors may play a role in this outcome.

CONCLUSION

1. Both weekly supplementation of Fe/Folate and Fe/Folate with Vitamin C group showed increase in mean hemoglobin levels in the anemic children.
2. Increment in Hb levels of subjects with addition of Vitamin C to weekly iron/folate supplementation was more effective than that with supplementation of iron/folate alone at the end of 12 and 24 weeks.
3. Prevalence of mild and moderate anemia decreased in both groups but Fe/Folate with Vitamin C showed dramatic response.
4. There is a gain in height in both groups but there is slightly increased gain noticed in added Vitamin C group at the end of 12 weeks.
5. Weight gain showed no difference in between groups.
6. Compliance is better in both groups and side effects are also minimal.
7. Nutrition education and IEC about anemia in schools is a must and to be integrated in these approaches to reduce the anemia effectively.
8. Weekly once Fe/Folate with Vitamin C supplementation showed better hemoglobin increment in short duration in the anemic children.
9. Suggested that a public health approach consisting of once weekly distribution of iron/folate with added Vitamin C through schools under supervision may be a better strategy than giving weekly iron/folate alone in adolescent girls to combat anemia.

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ANNEXURE – I

Data Collection Form

Name: Date:

Age:

Date of birth:

Sex:

Class:

School:

Group: A [weekly IFA only] / B [weekly IFA with Vitamin C]

Anthropometry: At Enrollment At 12 Weeks At 24 Weeks

Height (cm):

Weight (kg):

Body Mass Index:

Nutritional status: Undernutrition / Normal / Overweight
(Based On NCHS Standard)

Puberty Status:

Investigation:

	At Enrollment	12 Weeks	24 Weeks
Hb % in gm/dl			

ANNEXURE – II

Qualitative Data

1. How is the memory power of your child?

   Not satisfactory / Average / Good

2. Does your child is physically very active?

   Not satisfactory / Average / Good

3. Activities of your child in outdoor games?

   Not Satisfactory / Average / Good

4. How is your child in their studies?

   Not Satisfactory / Average / Good

5. How is the appetite of your child?

   Not Satisfactory / Average / Good

6. How is the sleep pattern of your child?

   Not Satisfactory / Average / Good

7. Does your child have frequent attacks of cough?

   Yes / Occasionally / No

8. Overall performance of your child?

   Not satisfactory / Average / good

Date:

Place: **Signature of parent**

ANNEXURE – III

Awareness Questionnaire about Anemia

1. Does anemia affect adolescent girls?

   Yes / No

2. Does worm infestation lead to nutritional anemia?

   Yes / No

3. Do you know about iron rich foods?

   Yes / No

4. Does tea intake affect absorption of iron?

   Yes / No

5. Does anemia cause cognitive impairment?

   Yes / No

6. Does anemia lead to delivery related complications in pregnant women?

   Yes / No

Name of the student:

Date:

Place:

ANNEXURE - IV

Cyanmethemoglobin (Hemoglobin cyanide) method

It is the WHO recommended method for determining the Hb concentration**.**

Basis of the method is dilution of blood in a solution containing potassium cyanide and potassium ferricyanide. Haemoglobin, methemoglobin, and carboxyhemoglobin but not sulphemoglobin are converted to HiCN. The absorbance of the solution is then measured in spectrometer at a wave length of 540nm or a photoelectric calorimeter with a yellow green filter.

Diluents:

Drabkins cyanide ferricyanide solution with PH of 7.0-7.4

Pottassium ferricyanide (0.607 mmol/l) 200mg

Potassium cyanide (0.768 mmol/l) 50mg

Pottassium dihydrogen phosphate (1.029 mmol/l) 140mg

Nonionic detergent 1ml

Distilled or deiodinised water 1 litre

Method

Make a 1 in 201 dilution of blood by adding 20ul of blood 5ml of diluents. After being allowed to stand at room temperature for at least 5 min, pour test sample into the cuvette and read the absorbance in a spectrophotometer at 540nm or in a photoelectric calorimeter.

Advantages

1) It allows the direct comparison with HiCN standard after dilution so batching is possible.

2) All forms of hemoglobin except sHb are readily converted to HiCN

ANNEXURE - V

WEEKLY ORAL IRON EFFICACY STUDY INFORMED CONSENT FORM – SCREENING

நடத்தும் முறை :

இந்த ஆய்வில் பங்கு கொள்வதற்கு உங்கள் குழந்தை 10 முதல் 14 வயதுடையவராக இருக்க வேண்டும். உங்கள் குழந்தை உடல் நலத்தை பரிசோதிக்க அவர்கள் முழு மருத்துவ குறிப்புகள் கேட்டறிந்து கொள்ளப்படும். முக்கியமாக உங்கள் குழந்தைக்கு இரத்த சோகைக்காக ஏதேனும் மருந்து உட் கொள்ளுதல், நீண்ட நாள் வியாதி, இரத்தப்போக்கு நோய் போன்றவை இருப்பின் இந்த ஆய்வுக்கு தகுதியில்லாதவராவர்.

மேலும் உங்கள் குழந்தை இந்த ஆய்வில் பங்கு கொள்ள வேண்டுமென்றால் இரத்தப் பரிசோதனை (Hb %) செய்ய வேண்டும். இந்த இரத்த பரிசோதனையில் உங்கள் குழந்தைக்கு Hb அளவு 7 gm / dl கீழே மிகவும் பாதிக்கப்பட்டிருந்தாலோ 12 gm / dl க்கு மேலே இருந்தாலோ ஆய்வில் பங்கு கொள்ள முடியாது. உங்கள் குழந்தையின் ஆய்வுகள் பற்றிய விவரங்களை தெரிந்து கொள்ள வேண்டுமென்றால் தயவு செய்து உங்களுடைய கேள்விகளை கேட்கவும். இரத்தத்தில் Hb அளவு 7 gm / dl குறைவாக இருப்பின் தகுந்த மருத்துவ ஆலோசனைகள் இலவசமாக வழங்கப்படும். இந்த பரிசோதனையில் உங்கள் குழந்தை பங்கு கொள்வதால் இரத்த சோகை உள்ளதா என்று தெரியவரும்.

இடர்கள் : இரத்தம் எடுக்கும் போது சிறிய வலி ஏற்படும்.

இவ்வாய்வில் பங்குபெறும் எல்லா நபர்களுடைய விபரங்களும் இரகசியமாகப் பாதுகாக்கப்படும். ஒழுக்கம் தொடர்பான குழுவினரும், இந்த ஆய்வை நடத்துகின்ற நபர்கள் மட்டும் உங்களுடைய குழந்தையின் குறிப்புகளை பார்வையிட அதிகாரம் உள்ளவர்கள். உங்கள் குழந்தை இந்த ஆய்வில் தொடர்ந்து இருக்க வேண்டும் என்பது கட்டாயமல்ல, எந்த நேரத்திலும் உங்கள் பங்களிப்பை நிறுத்திக் கொள்ளலாம். உங்கள் குழந்தை இந்த ஆய்வில் பங்கு கொள்ளவில்லை என்றாலோ அல்லது இடையில் நிறுத்திக் கொள்ள முடிவு செய்தாலோ அதற்காக நீங்கள் எந்தவித அபராதமும் கட்டத் தேவையில்லை.

உங்கள் குழந்தைக்கு செய்யப்படுகின்ற அனைத்து மருத்துவ பரிசோதனைகளும் இலவசமாக செய்யப்படும். இந்த தொடராய்வில் செய்யப்படுகின்ற செய்முறைகளினால் ஏற்படும் பக்க விளைவுகளுக்கு மருத்துவ உதவி செய்யப்படும். எந்தவித நஷ்ட ஈடும் தரப்பட மாட்டாது என்பதையும் அறிந்து கொண்டேன்.

1. நான் இந்த _____ தேதியிட்ட தகவல் படித்தை நன்றாகப் படித்து, படித்துக் காட்டி எடுத்துரைத்ததை புரிந்து கொண்டேன். எனக்கு கேள்வி கேட்கும் வாய்ப்பும் கிடைத்தது.
2. இந்த ஆய்வில் நான் என்னுடைய சுய அறிவோடு பங்கு கொள்கிறேன். மேலும் ஆய்விலிருந்து எந்த வித காரணமும் தராமல் மருத்துவப் பரிசோதனையிலிருந்து நான் விலகிக் கொள்ளலாம். இதற்கு சட்டரீதியான எந்த செயலும் உட்படுத்தாது.
3. Ethics குழுவின் அங்கத்தினர்களோ, இந்த ஆய்வை நடத்துபவர்களோ என்னுடைய மருத்துவ ஆய்வின் அனைத்து விவரங்களையும் என்னுடைய அனுமதியின்றி பார்க்கவோ, படிக்கவோ உரிமையுள்ளவர்களாவர். நான் இந்த ஆய்விலிருந்து விலகிக் கொண்டாலும் கூட என்னுடைய விவரங்களை அவர்கள் அறிந்து கொள்ள ஒத்துக் கொள்கிறேன். என்னுடைய விவரங்கள் அனைத்தும் 3 வது நபருக்கோ, அல்லது பத்திரிக்கையில் வெளியிடுவதற்கோ முயல மாட்டீர்கள் என நம்புகிறேன்.
4. இந்த ஆய்விலிருந்து பெறப்பட்ட புள்ளி விவரங்களையோ அல்லது முடிவுகளை பயன்படுத்த கூடாது என்று கட்டுப்படுத்த மாட்டேன்.
5. என் குழந்தையை இந்த மருத்துவ ஆய்விற்கு பங்கு கொள்ள பரிபூரணமாக சம்மதிக்கிறேன்.

கையொப்பம் _____
பெயர் _____
குழந்தையின் பெயர் _____

ANNEXURE - VI

WEEKLY ORAL IRON SUPPLEMENTATION EFFICACY STUDY

தெரிவிக்கப்பட்ட ஒப்புதல் படிவம்

நமது நாட்டில் ஏறக்குறைய 50% குழந்தைகளுக்கு இரத்த சோகை பாதிப்பு உள்ளது. இரும்பு சத்து குறைவே இதற்கு மிக முக்கிய காரணமாகும். குழந்தையின் அறிவு வளர்ச்சி, உடல் ஆரோக்கியம் மற்றும் நரம்பு மண்டல வளர்ச்சியில் இரத்த சோகை பாதிப்பை ஏற்படுத்த வல்லது. மேலும் இதனால் நோய் எதிர்ப்பு சக்தி குறைவு, உடலில் சோர்வு போன்ற பல உடல் நல குறைபாடுகள் ஏற்படும்.

இந்த ஆய்வின் மூலம் இரத்த சோகை மருத்துவத்திற்கு வாரம் ஒருமுறை இரும்பு சத்து மாத்திரை மற்றும் வைட்டமின் சி வழங்குதல் முறை மற்றும் வாரம் ஒரு முறை இரும்பு சத்து மாத்திரை மட்டும் வழங்குதல் முறை ஆகிய இரண்டு முறைகளையும் திறனாய்வு செய்யப்படும். இந்த ஆய்வின் மூலம் கிடைக்கப்பெறும் தகவல்களைக் கொண்டு இரத்த சோகை களைவதற்கான வழிமுறைகளை ஆராயப்படும்.

இந்த ஆய்வில் பங்கு கொள்ள தங்கள் வரவேற்கப்படுகிறீர்கள். இந்த ஆய்வின் பொருட்டு தங்கள் குழந்தையின் எடை, உயரம், இரத்த பரிசோதனை (Hb%) ஆகியவை ஆய்வின் ஆரம்ப நிலையிலும் 3, 6 மாத இறுதியிலும் மேற்கொள்ளப்படும். இதற்காக தங்கள் குழந்தையிடமிருந்து 0.02ml அளவு இரத்தம் தேவைப்படும்.

மருத்துவ ஆய்வு மற்றும் பரிசோதனையில் தங்கள் குழந்தைக்கு இரத்தசோகை (Hb 7-12 gm%) உள்ளது கண்டறியப்பட்டால் மட்டுமே இந்த ஆய்விற்கு தொடர்ந்து அனுமதிக்கப்படுவர். மேற்படி சோதனையில் தங்கள் குழந்தைக்கு இரத்த சோகை உள்ளது கண்டறியப்பட்டால் தொகுதி 1 (24 வாரங்களுக்கு வாரம் ஒரு முறை இரும்பு சத்து மாத்திரை மட்டும் பெறும் குழந்தைகள்) அல்லது தொகுதி 2 (6 வாரங்களுக்கு வாரம் ஒரு முறை இரும்பு சத்து மாத்திரை மற்றும் வைட்டமின் சி மாத்திரை பெறும் குழந்தைகள்) இவற்றில் ஒன்றில் வகுக்கப்பட்டு இரும்பு சத்து மாத்திரைகள் வழங்கப்படும். 12, 24 வார முடிவில் மீண்டும் ஒரு முறை மேற்கண்ட பரிசோதனைகள் தங்கள் குழந்தைகளுக்கு செய்யப்படும். இந்த ஆய்வில் தங்களின் பங்களிப்பால் மேலே குறிப்பிட்ட இரண்டு வகை மருத்துவ முறைகளை திறனாய்வு செய்ய வழவகுக்கும்.

இந்த ஆய்வில் பங்கேற்பதால் உங்கள் குழந்தைக்கு இரத்த சோகை உள்ளது கண்டறியப்பட்டு அதற்கான மருத்துவ சிகிச்சையும் இலவசமாக அளிக்கப்படும். பரிசோதனையில் தங்கள் குழந்தைக்கு ஏற்படும் கஷ்டங்கள் யாதெனில் இரத்தம் எடுக்கும் போது சிறு வலி மட்டுமே. இந்த ஆய்வில் உங்கள் குழந்தை பங்கேற்பது மனமுவந்து நீங்களாகவே முன்பு வந்தாலும், இந்த ஆராய்ச்சியில் கட்டாயம் தொடர்ந்து இருக்க வேண்டும் என்றில்லாமல், நீங்கள் எந்த நேரத்திலும் உங்கள் குழந்தையை இவ்வாய்வின் இருந்து விலகிக் கொள்ளலாம். நீங்கள் இந்த ஆராய்ச்சியில் இல்லை என்றாலோ அல்லது ஆய்விலிருந்து விலகிக் கொள்ளத் தீர்மானித்தாலோ அதற்காக உங்களிடமிருந்து நஷ்ட ஈடு எதுவும் கேட்கப்பட மாட்டாது.

பரிசோதனையாளர், உங்கள் குழந்தையை இந்த ஆய்வில் பங்கு பெறாமல் நிறுத்தும்படி கேட்கவும் வாய்ப்புள்ளது. இது எப்போது நடக்கும் எனில் நீங்கள் இந்த ஆய்வு நடக்கும் காலத்தில் வேறு ஏதாவது இரும்பு சத்து மருத்து எடுத்துக் கொண்டாலோ அல்லது முதன்மை பரிசோதனையாளர் இந்த ஆய்வில் தொடர்ந்து இருந்தால் குழந்தையின் உடல் நலத்திற்கு நல்லதல்ல என்று நம்பினாலும் நிறுத்தப்படுவீர்கள்.

Confidentiality : தானாக முன்வந்து பரிசோதனையில் பங்குபெறும் நபர்களின் விவரங்கள் அனைத்தும் இரகசியமாக பாதுகாக்கப்படும்.

உடல் பரிசோதனை மற்றும் இரத்தப்பரிசோதனை கட்டண எதுவுமின்றி இலவசமாக செய்யப்படும். ஆய்வு தொடங்கும் முன்பாகவோ அல்லது நடந்து கொண்டிருக்கும் சமயத்திலோ உங்களுக்கு ஏதாவது சந்தேகங்கள் இருந்தால் கேட்கலாம். அப்படி ஆய்வைப் பற்றிக் கேட்கும் கேள்விகள் மருத்துவரைக் கேட்கலாம்.

இந்த தொடராய்வில் செய்யப்படுகின்ற செய்முறைகளினால் ஏற்படும் பக்க விளைவுகளுக்கு மருத்துவ உதவி செய்யப்படும்.

1. நான் இந்த _____ தேதியிட்ட தகவல் படித்தை நன்றாகப் படித்து, படித்துக் காட்டி எடுத்துரைத்ததை புரிந்து கொண்டேன். எனக்கு கேள்வி கேட்கும் வாய்ப்பும் கிடைத்தது.
2. இந்த ஆய்வில் நான் என்னுடைய சுய அறிவோடு பங்கு கொள்கிறேன். மேலும் ஆய்விலிருந்து எந்த வித காரணமும் தராமல் மருத்துவப் பரிசோதனையிலிருந்து நான் விலகிக் கொள்ளலாம். இதற்கு சட்டரீதியான எந்த செயலும் உட்படுத்தாது.
3. Ethics குழுவின் அங்கத்தினர்களோ, இந்த ஆய்வை நடத்துபவர்களோ என்னுடைய மருத்துவ ஆய்வின் அனைத்து விவரங்களையும் என்னுடைய அனுமதியின்றி பார்க்கவோ, படிக்கவோ உரிமையுள்ளவர்களாவர். நான் இந்த ஆய்விலிருந்து விலகிக் கொண்டாலும் கூட என்னுடைய விவரங்களை அவர்கள் அறிந்து கொள்ள ஒத்துக் கொள்கிறேன். என்னுடைய விவரங்கள் அனைத்தும் 3 வது நபருக்கோ, அல்லது பத்திரிக்கையில் வெளியிடுவதற்கோ முயல மாட்டீர்கள் என நம்புகிறேன்.
4. இந்த ஆய்விலிருந்து பெறப்பட்ட புள்ளி விவரங்களையோ அல்லது முடிவுகளை பயன்படுத்த கூடாது என்று கட்டுப்படுத்த மாட்டேன்.
5. என் குழந்தையை இந்த மருத்துவ ஆய்விற்கு பங்கு கொள்ள பரிபூரணமாக சம்மதிக்கிறேன்.

கையொப்பம் _____

பெயர் _____

குழந்தையின் பெயர் _____

Signature of the Investigator : Date :

Signature of the Witnesses : Date :

The Dean

Govt. Kilpauk Medical College, Chennai-10

Sub: Ethical Committee project work - approved.

Ref: Lr.No.3944/P&D/09 Dt. 17.11.09.

With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 26.11.09.

1.	Dr.H.K.Fathima Prof & HOD of O & G	Hospital based surveillance of female Genital fistula.
2.	Dr.P.Thirunavukkarasu Asso. Prof of Physical Medicine	Coracoid Pain test – for adhesive capsulitis – a validation study.
3.	1. Dr.Praveen Kumar Dept of ENT 2. Dr.Venugopal 3. Dr.Karthik.V 4.Dr. Karthikha.R 5. Raghavi.N	Epidemiological study on Head and Neck Malignancies.
4.	Dr.S.Stalin, PG, MD (Paed)	Management of Anemia in Adolescent Girls.
5.	Dr.G.Kannan, PG in Phy Dept	A study of Serum lipid profile in Parkinson's Disease
6.	Dr.Adhira CRRI	A Prospective study of Demographic/Biological markers in suicide
7.	Dr.D.Sivalingam. PG MD(Psy)	A study of suicide attempt by self immolation, are the factors associated with it unique or universal.

We are glad to inform you that at the Ethical Committee meeting the documents were discussed and the above short term projects are Ethically approved.

CHAIRPERSON 21.12.09

Prof. Dr.V.KANAGASABAI, MD.,

The Dean

Govt. Kilpauk Medical College,

Chennai-10.

To

The Individuals

MASTER CHART - GROUP A (WEEKLY ONCE IRON / FOLIC ACID ONLY)

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
1	A	SHEELA	10	3		142	53	26.28	143.5	54	26.22	144.5	54	25.86	10.8	12.2	12.5
2	A	REVATHY	10	4		132	23	13.2	133.8	23.5	13.13	134.6	24	13.25	10.6	11	11.5
3	A	RUTHRA	10	3		131	22	12.82	133.4	24	13.49	133.9	25	13.94	11.3	12	12.3
4	A	A.PRIYA	10	4		121	20	13.66	123.7	22	14.38	124.8	22	14.13	10.4	11.6	12
5	A	SARATHA	10	4	y	145	33	15.7	145.5	34	16.06	147	35	16.2	11.5	12.5	12.5
6	A	JEGATHEESWARI	11	4		130	28	16.57	131.7	29	16.72	133	29	16.39	9.1	11.3	12
7	A	V.NANDHINI	11	4		126	22	13.86	127.3	23	14.19	128.5	23.5	14.23	10.2	11	12
8	A	SHIFANA	11	3		134.5	26	14.37	136	27	14.6	137.3	28	14.85	11.4	12	13
9	A	SANDHYA	11	4		131	30	17.48	132.8	32	18.14	133.8	33	18.43	11.2	12.2	12.8
10	A	LAKSHMI	12	4		132	26	14.92	134	27	15.04	135.5	27.5	14.98	11.4	11.5	12.4
11	A	ABIRAMI	11	3		134	25	13.92	136	26	14.06	137.5	27	14.28	10.7	12.2	12.5
12	A	A.NANDHINI	11	3		126	23	14.49	127.4	24	14.79	127.6	24.5	15.05	11.5	12	12.5
13	A	MUTHALAGHI	11	4		138.7	25	13	139.2	25	12.9	140	26	13.27	10.8	11.8	12.3
14	A	GIRIJA	12	4		144	36	17.37	145.8	37	17.41	146.5	37.5	17.47	10.7	11.7	12.4
15	A	KUZHALI	13	3		144	32	15.43	145.8	34	15.99	146	34.5	16.19	9 .1	10.5	11
16	A	KALAISELVI	14	3	y	154	43	18.13	156.2	45	18.44	157.1	45.5	18.44	11.5	12	12
17	A	SATHYA	13	3	y	147	33	15.27	147.6	35	16.07	148	35	15.98	10.7	11.5	11.3
18	A	REKHA	13	3	y	151	41	17.98	151.6	41.5	18.06	151.8	42	18.23	10.8	11	11.5
19	A	SHANTHI	12	4	y	144	40	19.29	145.5	41	19.37	146	41	19.23	10.2	11.5	12
20	A	SELVI	12	4		140	29	14.8	141	30	15.09	142	30	14.88	10.8	12	12
21	A	SRIPRIYA	12	4		131	31	18.06	132	33	18.94	133.5	33.5	18.8	10.7	11.5	12
22	A	YASODHA	12	4	y	150	47	20.89	151.2	49	21.43	151.5	49.5	21.57	9.8	10	12
23	A	RAMYA	11	4		133	27	15.26	134.7	29	15.98	136	30	16.22	8.6	9.2	9.8
24	A	NEELU	11	4		135	30	16.46	136.5	33	17.71	137.3	33.5	17.77	10.8	12	12.8
25	A	MAHALAKSHMI	12	4	y	139	25	12.94	141	28	14.08	141.5	28.5	14.23	11	12.2	12.8
26	A	RAJI	12	3	y	153	40	17.09	154	40.5	17.08	155	41	17.07	10.8	12	12.8
27	A	S.NANDHINI	11	4		136	29	15.68	137.9	31	16.3	139	31.5	16.3	10.8	12	12.5
28	A	GAYATHRI	12	4		134	26	14.48	136.4	28	15.05	137.2	29	15.41	10.7	12	12.2
29	A	ANJALI	13	4		139	28	14.49	140	29	14.8	140.8	30	15.13	11.5	12	12.5
30	A	GIRIJA	12	4	y	141	29	14.59	142.3	29	14.32	143.5	30	14.57	10.6	11.8	12.5

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
31	A	MONISHA	12	4		130	21	12.43	131.2	23.5	13.65	132.5	24	13.67	11	12	12.5
32	A	R.NANDHINI	12	4	y	147	35	16.2	149.5	36	16.11	150	37	16.44	10.4	11.5	11.7
33	A	NITHYA	12	4		141	27	13.6	142.2	29.5	14.59	143.5	29	14.08	11.5	12	12.5
34	A	VANAJA	13	3	y	148.5	35.5	16.1	149.5	37	16.55	150	37.5	16.67	10.8	11.8	12.6
35	A	SUDHA	12	5		142	30	14.9	143.1	31	15.14	144	31	14.95	8.5	10.4	11
36	A	ASHAVANI	13	4		140	31	15.8	141.1	33	16.58	142	33	16.37	11	11.8	12.4
37	A	MOHANAPRIYA	12	4	y	144	31	14.95	145.4	32	15.14	146.5	32.5	15.14	10.7	11.8	12.5
38	A	SUJATHA	13	5		143	29	14.2	145.5	31	14.64	146	32	15.01	8.1	9	9.8
39	A	SARITHA	12	5		144	35	16.9	145.2	36	17.08	145.8	36	16.94	8.6	10.2	11.2
40	A	DEEPA	13	5	y	136	24	13	136.8	25	13.36	137.8	26	13.69	7.2	8.2	9.2
41	A	NATHIYA	13	5	y	153	39	16.7	154.2	40	16.82	154.8	41	17.11	8.0	8.9	9.8
42	A	KANJAMMAL	13	4	y	151	34	14.9	152	35	15.15	153	35.5	15.17	9.2	9.9	10.6
43	A	PRABHAVATHI	12	3	y	152	42	18.2	152.6	42.5	18.25	153.5	43	18.25	10.7	11.8	12.2
44	A	PARIMALA SUJATHA	12	4		152	37	16	153	38	16.23	153.5	39	16.55	10.5	11.3	11.9
45	A	ANANDHI	14	3		150	36	16	151	37	16.23	151.5	38	16.56	10.5	11.2	11.9
46	A	DIVYA	13	4		147	35	16.2	147.5	37	17.01	148.5	38	17.23	8.2	9.4	10.4
47	A	JANAKI	14	4		153	42	17.9	154	43	18.13	154.5	43.5	18.22	10.7	11.5	12.2
48	A	LATHA	13	4	y	163	70	26.4	164	71	26.4	165	72	26.45	9.5	10.5	11.6
49	A	NISHAKUMARI	13	5		143	32	15.65	143.5	33	16.03	144	34	16.4	8.1	9.6	10.6
50	A	PRIYANKA	12	5		136	29	15.7	137.5	30	15.87	138	31	16.28	8.6	9.4	10.2
51	A	PREMA	13	4		137.5	25	13.22	139.3	26	13.4	140.8	27	13.62	8	9.2	10.2
52	A	SAVITHRI	13	5		130	20	11.8	131.5	22	12.72	132.5	22.5	12.82	10.7	11.5	12.2
53	A	USHA	12	3		145	39	18.55	147	41	18.97	148	41.5	18.95	11	11.8	12.5
54	A	UMA	13	4		132.5	26	14.81	134.2	27	14.99	135.4	27.5	15	9.6	10	11.2
55	A	VICITHRA	14	5	y	151	34	14.91	152.2	35	15.11	153	36	15.38	8	9.2	10.2
56	A	VARALAKSHMI	13	4		130	28	16.57	131.8	29	16.69	132.8	30	17.01	8.5	9.5	10.2
57	A	YUVARANI	13	5		138.5	27	14.08	140	28	14.29	141	29	14.59	8.6	9.6	10.8
58	A	POOVARASI	13	4		152	36	15.58	153.5	37	15.7	154.5	38	15.92	10.9	11.6	12.4

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
59	A	RISWANA	13	3	y	151.5	36	15.68	153.5	38	16.13	154.5	39	16.34	7.9	9.1	10.2
60	A	PRIYA	13	4		148	36	16.44	149.2	37	16.62	150	38	16.89	10.5	11.2	12.2
61	A	LAVANYA	14	4		146	33	15.48	147.8	34.5	15.79	148.5	35.5	16.1	10.8	11.8	12.4
62	A	DHARANI PRIYA	13	3		149	37	16.67	150	39	17.33	151	40	17.54	11.6	12.2	12.5
63	A	UMAMAHESWARI	14	3		145	44	20.93	146	45	21.11	146.8	45.5	21.11	9.8	10.8	11.5
64	A	PRIYA	14	4		149	35	15.77	150	36	16	151	36.5	16.01	10.7	11.7	12.5
65	A	NAGALAKSHMI	13	4	y	146.5	32	14.91	147.1	34	15.71	147.9	35	16	10.6	11.8	12.5
66	A	KEERTHANA	13	4	y	153	43	18.37	153.8	44	18.6	154.5	45	18.85	11	11.8	12.5
67	A	VINODHINI	13	5	y	141	34	17.1	142.5	36	17.73	143.5	36.5	17.73	7.8	9.2	9.9
68	A	LAVANYA	14	5		145	39	18.55	145.3	40	18.95	146	40	18.77	7.6	8.8	9.9
69	A	PARAMESWARI	13	4		151	32	14.03	152.3	34.5	14.87	153.6	35	14.83	10.8	11.8	12.5
70	A	JEEVITHA	12	4		141.5	27	13.48	142.9	28.5	13.96	143.9	29	14	10.7	11.8	12.5
71	A	DEEPA	14	4	y	152	51	22.07	152.3	52	22.42	153	52.5	22.43	11.4	11.8	12.3
72	A	SIVAGAMI	14	4		158	37	14.82	158.7	37	14.69	159.6	37.5	14.72	10.7	12.0	12.5
73	A	JESI	14	4		158	48	19.23	159	49	19.38	159.5	49.5	19.46	11.2	11.9	12.5
74	A	PAVITHRA	13	4		135	27	14.81	136.2	27.5	14.82	137	28	14.92	11.2	11.8	12.4
75	A	JEYACHITHRA	12	4		146	29	13.6	146.9	30	13.9	147.6	31	14.23	10.8	12	12.5
76	A	VANITHA	13	4	y	142.5	43	21.18	143	44	21.52	143.5	44.5	21.61	11.6	12.2	12.8
77	A	HAJEERA	11	3		138	23	12.08	139.8	24.5	12.54	140.5	25.5	12.92	11	11.8	12.5
78	A	RESHMA	11	4		129	26	15.62	130.2	27	15.93	130.5	27.5	16.15	10.2	11.3	11.8
79	A	NAJMA	11	3		130	25	14.79	131.4	26	15.06	131.7	27	15.57	8	9	9.8
80	A	MUDHARSANA BEGUM	11	4		135	22	12.07	136.7	23	12.31	137	24	12.79	11	11.5	12
81	A	AKBAR NISHA	11	4		138	30	15.75	139.6	32	16.42	140.5	33	16.72	10.7	11	11.5
82	A	AHMED SHERIN	11	4		143	29	14.18	144.4	31	14.87	145.2	32	15.18	11	11.5	12
83	A	ARCHANA	12	3		136	30	16.22	138	32	16.8	139	33	17.08	11	12	12
84	A	JABENA	12	4		145	38	18.07	146.4	39	18.2	147	39	18.05	11	12	12.5
85	A	ARIFA BEGUM	12	3	y	143	29	14.18	143.5	31	15.05	144	31	14.95	10.7	11.7	12.5

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
86	A	AMMU	12	4	y	147	45	20.82	148	46	21	148.5	47	21.31	11	11.7	12.4
87	A	A.SUBASHINI	13	4	y	151	39	17.1	152	40	17.31	152.5	40.5	17.41	11	12	12.4
88	A	R.AJISHA	13	4		139	33	17.08	141.2	34	17.05	142	35	17.36	11	12	12.5
89	A	THAIYABA	13	4		145	31	14.74	147	31.5	14.58	147.5	32	14.71	9.9	11.4	12.2
90	A	A.SHAKIN	12	4		140.5	32	16.21	142.4	33.5	16.52	143.4	34	16.53	11	11.5	12
91	A	M.SHAINAZ	13	4	y	147	35	16.2	147.7	36.5	16.73	148	37.5	17.12	9.5	10.5	11
92	A	B.SIMRAN	13	4	y	156	42	17.26	157.1	43	17.42	158	44	17.63	11	12	12.5
93	A	L.NAAZIABEGUM	14	4	y	156	33	13.56	158.6	35	13.91	159.5	36	14.15	11	11.8	12.5
94	A	P.PARVEEN BANU	14	4	y	147	39.5	18.28	149	41	18.47	149.4	42	18.82	11	12	12.5
95	A	H.GOWSIA BEGUM	14	4	y	157	59	23.94	158	59.5	23.83	158.5	60	23.88	11	12	12.5
96	A	M.MEENA	13	4	y	156.5	37	15.11	158	39.5	15.82	158.5	40	15.92	11	11.6	12.5
97	A	R.SHALINI	13	4	y	146.5	51	23.76	147.1	52	24.03	147.4	53	24.39	11	11.6	12.5
98	A	SALMA BEGUM	12	4		143	25	12.23	144	25	12.06	144.5	26	12.45	11	12	12.5
99	A	E.AMRIN BEGUM	13	4		144	30	14.47	146.2	31	14.5	146.8	32	14.85	11	12	11
100	A	K.SHAKILA BANU	13	4	y	150	37	16.44	151.1	38	16.64	151.5	39	16.99	11	11.5	12
101	A	F.FOUSIYA	14	3	y	143	52	25.43	144	53	25.56	144.6	54	25.83	10.8	11	11.8
102	A	E.DEEPA	13	4	y	144	43	20.74	145	44	20.93	145.5	44.5	21.02	11	11.8	11.9
103	A	RAMYA	13	4		133	25	14.13	134	26	14.48	135.5	26.5	14.43	8	9	9.7
104	A	NAJMA	13	4	y	143	25	12.23	144	25	12.06	144.5	26	12.45	9	10.4	11.2
105	A	FATHIMA	13	4	y	136	30	16.22	137	31	16.52	138.5	32	16.68	7.6	8.2	9.6
106	A	A.THASLEEM	12	3		143	52	25.43	144	53	25.56	144.6	54	25.83	10.8		
107	A	C.UMA	13	4		144	43	20.74	145	44	20.93	145.5	44.5	21.02	11.2		
108	A	SARASWATHI	13	4		133	25	14.13	134	26	14.48	135.5	26.5	14.43	11.3		
109	A	PARVEEN BEGUM	14	4		143	25	12.23	144	25	12.06	144.5	26	12.45	9		
110	A	KUMARI	14	4		136	30	16.22	137	31	16.52	138.5	32	16.68	10.7		

MASTER CHART - GROUP A (WEEKLY ONCE IRON / FOLIC ACID ONLY)

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
1	A	SHEELA	10	3		142	53	26.28	143.5	54	26.22	144.5	54	25.86	10.8	12.2	12.5
2	A	REVATHY	10	4		132	23	13.2	133.8	23.5	13.13	134.6	24	13.25	10.6	11	11.5
3	A	RUTHRA	10	3		131	22	12.82	133.4	24	13.49	133.9	25	13.94	11.3	12	12.3
4	A	A.PRIYA	10	4		121	20	13.66	123.7	22	14.38	124.8	22	14.13	10.4	11.6	12
5	A	SARATHA	10	4	y	145	33	15.7	145.5	34	16.06	147	35	16.2	11.5	12.5	12.5
6	A	JEGATHEESWARI	11	4		130	28	16.57	131.7	29	16.72	133	29	16.39	9.1	11.3	12
7	A	V.NANDHINI	11	4		126	22	13.86	127.3	23	14.19	128.5	23.5	14.23	10.2	11	12
8	A	SHIFANA	11	3		134.5	26	14.37	136	27	14.6	137.3	28	14.85	11.4	12	13
9	A	SANDHYA	11	4		131	30	17.48	132.8	32	18.14	133.8	33	18.43	11.2	12.2	12.8
10	A	LAKSHMI	12	4		132	26	14.92	134	27	15.04	135.5	27.5	14.98	11.4	11.5	12.4
11	A	ABIRAMI	11	3		134	25	13.92	136	26	14.06	137.5	27	14.28	10.7	12.2	12.5
12	A	A.NANDHINI	11	3		126	23	14.49	127.4	24	14.79	127.6	24.5	15.05	11.5	12	12.5
13	A	MUTHALAGHI	11	4		138.7	25	13	139.2	25	12.9	140	26	13.27	10.8	11.8	12.3
14	A	GIRIJA	12	4		144	36	17.37	145.8	37	17.41	146.5	37.5	17.47	10.7	11.7	12.4
15	A	KUZHALI	13	3		144	32	15.43	145.8	34	15.99	146	34.5	16.19	9 .1	10.5	11
16	A	KALAISELVI	14	3	y	154	43	18.13	156.2	45	18.44	157.1	45.5	18.44	11.5	12	12
17	A	SATHYA	13	3	y	147	33	15.27	147.6	35	16.07	148	35	15.98	10.7	11.5	11.3
18	A	REKHA	13	3	y	151	41	17.98	151.6	41.5	18.06	151.8	42	18.23	10.8	11	11.5
19	A	SHANTHI	12	4	y	144	40	19.29	145.5	41	19.37	146	41	19.23	10.2	11.5	12
20	A	SELVI	12	4		140	29	14.8	141	30	15.09	142	30	14.88	10.8	12	12
21	A	SRIPRIYA	12	4		131	31	18.06	132	33	18.94	133.5	33.5	18.8	10.7	11.5	12
22	A	YASODHA	12	4	y	150	47	20.89	151.2	49	21.43	151.5	49.5	21.57	9.8	10	12
23	A	RAMYA	11	4		133	27	15.26	134.7	29	15.98	136	30	16.22	8.6	9.2	9.8
24	A	NEELU	11	4		135	30	16.46	136.5	33	17.71	137.3	33.5	17.77	10.8	12	12.8
25	A	MAHALAKSHMI	12	4	y	139	25	12.94	141	28	14.08	141.5	28.5	14.23	11	12.2	12.8
26	A	RAJI	12	3	y	153	40	17.09	154	40.5	17.08	155	41	17.07	10.8	12	12.8
27	A	S.NANDHINI	11	4		136	29	15.68	137.9	31	16.3	139	31.5	16.3	10.8	12	12.5
28	A	GAYATHRI	12	4		134	26	14.48	136.4	28	15.05	137.2	29	15.41	10.7	12	12.2
29	A	ANJALI	13	4		139	28	14.49	140	29	14.8	140.8	30	15.13	11.5	12	12.5
30	A	GIRIJA	12	4	y	141	29	14.59	142.3	29	14.32	143.5	30	14.57	10.6	11.8	12.5

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
31	A	MONISHA	12	4		130	21	12.43	131.2	23.5	13.65	132.5	24	13.67	11	12	12.5
32	A	R.NANDHINI	12	4	y	147	35	16.2	149.5	36	16.11	150	37	16.44	10.4	11.5	11.7
33	A	NITHYA	12	4		141	27	13.6	142.2	29.5	14.59	143.5	29	14.08	11.5	12	12.5
34	A	VANAJA	13	3	y	148.5	35.5	16.1	149.5	37	16.55	150	37.5	16.67	10.8	11.8	12.6
35	A	SUDHA	12	5		142	30	14.9	143.1	31	15.14	144	31	14.95	8.5	10.4	11
36	A	ASHAVANI	13	4		140	31	15.8	141.1	33	16.58	142	33	16.37	11	11.8	12.4
37	A	MOHANAPRIYA	12	4	y	144	31	14.95	145.4	32	15.14	146.5	32.5	15.14	10.7	11.8	12.5
38	A	SUJATHA	13	5		143	29	14.2	145.5	31	14.64	146	32	15.01	8.1	9	9.8
39	A	SARITHA	12	5		144	35	16.9	145.2	36	17.08	145.8	36	16.94	8.6	10.2	11.2
40	A	DEEPA	13	5	y	136	24	13	136.8	25	13.36	137.8	26	13.69	7.2	8.2	9.2
41	A	NATHIYA	13	5	y	153	39	16.7	154.2	40	16.82	154.8	41	17.11	8.0	8.9	9.8
42	A	KANJAMMAL	13	4	y	151	34	14.9	152	35	15.15	153	35.5	15.17	9.2	9.9	10.6
43	A	PRABHAVATHI	12	3	y	152	42	18.2	152.6	42.5	18.25	153.5	43	18.25	10.7	11.8	12.2
44	A	PARIMALA SUJATHA	12	4		152	37	16	153	38	16.23	153.5	39	16.55	10.5	11.3	11.9
45	A	ANANDHI	14	3		150	36	16	151	37	16.23	151.5	38	16.56	10.5	11.2	11.9
46	A	DIVYA	13	4		147	35	16.2	147.5	37	17.01	148.5	38	17.23	8.2	9.4	10.4
47	A	JANAKI	14	4		153	42	17.9	154	43	18.13	154.5	43.5	18.22	10.7	11.5	12.2
48	A	LATHA	13	4	y	163	70	26.4	164	71	26.4	165	72	26.45	9.5	10.5	11.6
49	A	NISHAKUMARI	13	5		143	32	15.65	143.5	33	16.03	144	34	16.4	8.1	9.6	10.6
50	A	PRIYANKA	12	5		136	29	15.7	137.5	30	15.87	138	31	16.28	8.6	9.4	10.2
51	A	PREMA	13	4		137.5	25	13.22	139.3	26	13.4	140.8	27	13.62	8	9.2	10.2
52	A	SAVITHRI	13	5		130	20	11.8	131.5	22	12.72	132.5	22.5	12.82	10.7	11.5	12.2
53	A	USHA	12	3		145	39	18.55	147	41	18.97	148	41.5	18.95	11	11.8	12.5
54	A	UMA	13	4		132.5	26	14.81	134.2	27	14.99	135.4	27.5	15	9.6	10	11.2
55	A	VICITHRA	14	5	y	151	34	14.91	152.2	35	15.11	153	36	15.38	8	9.2	10.2
56	A	VARALAKSHMI	13	4		130	28	16.57	131.8	29	16.69	132.8	30	17.01	8.5	9.5	10.2
57	A	YUVARANI	13	5		138.5	27	14.08	140	28	14.29	141	29	14.59	8.6	9.6	10.8
58	A	POOVARASI	13	4		152	36	15.58	153.5	37	15.7	154.5	38	15.92	10.9	11.6	12.4

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
59	A	RISWANA	13	3	y	151.5	36	15.68	153.5	38	16.13	154.5	39	16.34	7.9	9.1	10.2
60	A	PRIYA	13	4		148	36	16.44	149.2	37	16.62	150	38	16.89	10.5	11.2	12.2
61	A	LAVANYA	14	4		146	33	15.48	147.8	34.5	15.79	148.5	35.5	16.1	10.8	11.8	12.4
62	A	DHARANI PRIYA	13	3		149	37	16.67	150	39	17.33	151	40	17.54	11.6	12.2	12.5
63	A	UMAMAHESWARI	14	3		145	44	20.93	146	45	21.11	146.8	45.5	21.11	9.8	10.8	11.5
64	A	PRIYA	14	4		149	35	15.77	150	36	16	151	36.5	16.01	10.7	11.7	12.5
65	A	NAGALAKSHMI	13	4	y	146.5	32	14.91	147.1	34	15.71	147.9	35	16	10.6	11.8	12.5
66	A	KEERTHANA	13	4	y	153	43	18.37	153.8	44	18.6	154.5	45	18.85	11	11.8	12.5
67	A	VINODHINI	13	5	y	141	34	17.1	142.5	36	17.73	143.5	36.5	17.73	7.8	9.2	9.9
68	A	LAVANYA	14	5		145	39	18.55	145.3	40	18.95	146	40	18.77	7.6	8.8	9.9
69	A	PARAMESWARI	13	4		151	32	14.03	152.3	34.5	14.87	153.6	35	14.83	10.8	11.8	12.5
70	A	JEEVITHA	12	4		141.5	27	13.48	142.9	28.5	13.96	143.9	29	14	10.7	11.8	12.5
71	A	DEEPA	14	4	y	152	51	22.07	152.3	52	22.42	153	52.5	22.43	11.4	11.8	12.3
72	A	SIVAGAMI	14	4		158	37	14.82	158.7	37	14.69	159.6	37.5	14.72	10.7	12.0	12.5
73	A	JESI	14	4		158	48	19.23	159	49	19.38	159.5	49.5	19.46	11.2	11.9	12.5
74	A	PAVITHRA	13	4		135	27	14.81	136.2	27.5	14.82	137	28	14.92	11.2	11.8	12.4
75	A	JEYACHITHRA	12	4		146	29	13.6	146.9	30	13.9	147.6	31	14.23	10.8	12	12.5
76	A	VANITHA	13	4	y	142.5	43	21.18	143	44	21.52	143.5	44.5	21.61	11.6	12.2	12.8
77	A	HAJEERA	11	3		138	23	12.08	139.8	24.5	12.54	140.5	25.5	12.92	11	11.8	12.5
78	A	RESHMA	11	4		129	26	15.62	130.2	27	15.93	130.5	27.5	16.15	10.2	11.3	11.8
79	A	NAJMA	11	3		130	25	14.79	131.4	26	15.06	131.7	27	15.57	8	9	9.8
80	A	MUDHARSANA BEGUM	11	4		135	22	12.07	136.7	23	12.31	137	24	12.79	11	11.5	12
81	A	AKBAR NISHA	11	4		138	30	15.75	139.6	32	16.42	140.5	33	16.72	10.7	11	11.5
82	A	AHMED SHERIN	11	4		143	29	14.18	144.4	31	14.87	145.2	32	15.18	11	11.5	12
83	A	ARCHANA	12	3		136	30	16.22	138	32	16.8	139	33	17.08	11	12	12
84	A	JABENA	12	4		145	38	18.07	146.4	39	18.2	147	39	18.05	11	12	12.5
85	A	ARIFA BEGUM	12	3	y	143	29	14.18	143.5	31	15.05	144	31	14.95	10.7	11.7	12.5

SL.NO	GROUP	NAME	AGE	S.E.STATUS	PUB.ATTA	HT1	WT1	BMI 1	HT2	WT2	BMI2	HT3	WT3	BMI3	HB1	HB2	HB3
86	A	AMMU	12	4	y	147	45	20.82	148	46	21	148.5	47	21.31	11	11.7	12.4
87	A	A.SUBASHINI	13	4	y	151	39	17.1	152	40	17.31	152.5	40.5	17.41	11	12	12.4
88	A	R.AJISHA	13	4		139	33	17.08	141.2	34	17.05	142	35	17.36	11	12	12.5
89	A	THAIYABA	13	4		145	31	14.74	147	31.5	14.58	147.5	32	14.71	9.9	11.4	12.2
90	A	A.SHAKIN	12	4		140.5	32	16.21	142.4	33.5	16.52	143.4	34	16.53	11	11.5	12
91	A	M.SHAINAZ	13	4	y	147	35	16.2	147.7	36.5	16.73	148	37.5	17.12	9.5	10.5	11
92	A	B.SIMRAN	13	4	y	156	42	17.26	157.1	43	17.42	158	44	17.63	11	12	12.5
93	A	L.NAAZIABEGUM	14	4	y	156	33	13.56	158.6	35	13.91	159.5	36	14.15	11	11.8	12.5
94	A	P.PARVEEN BANU	14	4	y	147	39.5	18.28	149	41	18.47	149.4	42	18.82	11	12	12.5
95	A	H.GOWSIA BEGUM	14	4	y	157	59	23.94	158	59.5	23.83	158.5	60	23.88	11	12	12.5
96	A	M.MEENA	13	4	y	156.5	37	15.11	158	39.5	15.82	158.5	40	15.92	11	11.6	12.5
97	A	R.SHALINI	13	4	y	146.5	51	23.76	147.1	52	24.03	147.4	53	24.39	11	11.6	12.5
98	A	SALMA BEGUM	12	4		143	25	12.23	144	25	12.06	144.5	26	12.45	11	12	12.5
99	A	E.AMRIN BEGUM	13	4		144	30	14.47	146.2	31	14.5	146.8	32	14.85	11	12	11
100	A	K.SHAKILA BANU	13	4	y	150	37	16.44	151.1	38	16.64	151.5	39	16.99	11	11.5	12
101	A	F.FOUSIYA	14	3	y	143	52	25.43	144	53	25.56	144.6	54	25.83	10.8	11	11.8
102	A	E.DEEPA	13	4	y	144	43	20.74	145	44	20.93	145.5	44.5	21.02	11	11.8	11.9
103	A	RAMYA	13	4		133	25	14.13	134	26	14.48	135.5	26.5	14.43	8	9	9.7
104	A	NAJMA	13	4	y	143	25	12.23	144	25	12.06	144.5	26	12.45	9	10.4	11.2
105	A	FATHIMA	13	4	y	136	30	16.22	137	31	16.52	138.5	32	16.68	7.6	8.2	9.6
106	A	A.THASLEEM	12	3		143	52	25.43	144	53	25.56	144.6	54	25.83	10.8		
107	A	C.UMA	13	4		144	43	20.74	145	44	20.93	145.5	44.5	21.02	11.2		
108	A	SARASWATHI	13	4		133	25	14.13	134	26	14.48	135.5	26.5	14.43	11.3		
109	A	PARVEEN BEGUM	14	4		143	25	12.23	144	25	12.06	144.5	26	12.45	9		
110	A	KUMARI	14	4		136	30	16.22	137	31	16.52	138.5	32	16.68	10.7		